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27 Melville Square
July 11th 1895.

Pentland's Medical Series.

VOLUME SECOND.

DISEASES OF THE EYE.

DISEASES

OF

THE EYE.

A PRACTICAL TREATISE FOR STUDENTS OF OPHTHALMOLOGY

BY

GEORGE A. BERRY, M.B., F.R.C.S.Ed.,

OPHTHALMIC SURGEON, EDINBURGH ROYAL INFIRMARY; SENIOR SURGEON,
EDINBURGH EYE DISPENSARY; LECTURER ON OPHTHALMOLOGY,
ROYAL COLLEGE OF SURGEONS, EDINBURGH.

SECOND EDITION, REVISED AND ENLARGED.
WITH COLOURED ILLUSTRATIONS FROM ORIGINAL DRAWINGS.

EDINBURGH AND LONDON:
YOUNG J. PENTLAND.
1893.

EDINBURGH: PRINTED FOR YOUNG J. PENTLAND, TEVIOT PLACE, AND 38 WEST
SMITHFIELD, LONDON, E.C., BY SCOTT AND FERGUSON AND BURNESS AND COMPANY.

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TO

EDMUND HANSEN GRUT, M.D.,

PROFESSOR OF OPHTHALMOLOGY IN THE UNIVERSITY OF COPENHAGEN,

WHO, UNSURPASSED IN SKILL AS AN OPERATOR,

MAKES HIS LARGE EXPERIENCE EXCEPTIONALLY VALUABLE

BY BRINGING TO BEAR ON IT THE POWERS OF A

CRITICAL AND PRACTICAL MIND,

AND

TO WHOM, OF MANY TEACHERS IN OPHTHALMOLOGY,

THE AUTHOR IS MOST INDEBTED FOR ADVICE AND ASSISTANCE,

THIS BOOK IS AFFECTIONATELY DEDICATED.

PREFACE TO SECOND EDITION.

VERY great changes have been made in the present edition as regards both the matter and arrangement of the chapters. By these changes many of the more obvious defects of the former edition have, I hope, been removed. I have endeavoured in every way to maintain the practical character of the book; and for this purpose a large number of new drawings and diagrams have been introduced. The great amount of revision required, and the length of time involved in passing an illustrated work of this kind through the press, must be my apology for the delay in the appearance of a new edition.

My best thanks are due to Dr. Maddox for much valuable assistance in connection with the revision and correction of the proof sheets. I have also to thank Mr. Sydney Stephenson for several of the drawings illustrative of normal conditions of the back of the eye.

31 DRUMSHEUGH GARDENS,
EDINBURGH, *March* 1893.

PREFACE TO FIRST EDITION.

In the following pages I have endeavoured to give a description of the principal Diseases which affect the Eye, or lead in any way to impaired vision. The symptoms and treatment of these diseases are discussed more or less fully, according to their importance. Except in cases where it has some direct bearing on the treatment to be adopted, I have either altogether omitted any mention of Pathological Anatomy, or have devoted a relatively small space to it. This I have done for various reasons,—the main one being that I believe any exhaustive discussion of the Pathological Anatomy of Eye Diseases tends to divert attention from their clinical aspects. The objective examination of the eye, and the subjective examination of its functions, are capable of affording sufficient information for all practical purposes. The clinical study can therefore very well be carried on without much attention to the details of Pathology.

In Section I. of this work the basis of descriptions given is entirely clinical. The arrangement of chapters is perhaps somewhat different from that usually followed in similar treatises. Thus, separate chapters have been devoted to "Foreign Bodies in the Eye," "Sympathetic Ophthalmitis," and "Intraocular Tumours." The two former are subjects of so great practical importance that I have thought it right thus to emphasise them; all the more that they have frequently not received adequate notice in text-books. Intraocular Tumours, though of comparatively rare occurrence, are interesting to most Ophthalmic Surgeons, and the subject has received sufficient attention to fall naturally under a special chapter.

In Section II., which includes the chapters on Refraction, on the Ocular Muscles, and on the Methods of examining the Eye, the subjects have been treated in a manner which may be looked upon by some as not purely practical. One of the most essential parts of the work of an Ophthalmic Surgeon consists in the diagnosis and treatment of optical errors, and to fit him for this work a special theoretical knowledge of Refraction is indispensable. An accurate knowledge, too, of this branch of Ophthalmology is of the utmost importance to him—not so much in order that he may be able to correct errors of Refraction which are supposed to have some bearing on more general and obscure diseases—a connection, unintentionally no doubt, greatly exaggerated—but in order that, in the consideration of any existing visual defect, he may be able to eliminate the possible factor of optical error. As regards the chapter on the Ocular Muscles, the subject is one which is usually treated in rather a cursory manner. Anything like a full discussion of it necessarily involves a certain amount of speculation. The more theoretical portions are therefore, like the explanatory portions in the preceding and following chapters, printed in smaller type.

In order to avoid repetition, and to facilitate reference to Operations which are performed for several different affections of the Eye, all the principal Operations are discussed in the last chapter, which constitutes Section III.

For a knowledge of much that has been of the greatest use to me in the practice of my profession, I am indebted to Professor

Hansen Grut of Copenhagen. His teaching has, I feel, largely influenced me in the treatment of many of the following chapters, and I have particularly to thank him for a personal revision of some of the chapters, and for many valuable suggestions in connection with them, of which I have availed myself.

To Dr. Charnley of Shrewsbury my best thanks are due for revising the chapter on Refraction, and verifying the formulæ contained in it. Mr. Gunn, too, has kindly looked over the chapters on the Diseases of the Choroid and Retina. I have further to thank Drs. Symington and Hill Griffith for the loan of anatomical specimens from which some of the drawings are taken, and my former clinical assistant, Dr. Scott, for assisting me in reading the proofs. All the coloured drawings have been made from life by Dr. Tatham Thompson of Cardiff. They will, I hope, prove useful to those who may not be in the way of seeing a number of actual cases. Owing to Dr. Tatham Thompson having left Edinburgh before he had had time to finish the series of drawings, I have not been able personally to select all the cases represented. I cannot therefore claim for them that they are altogether as illustrative as might have been desirable. They are, however, truthful, and at the same time artistic representations of actual cases.

During the preparation of this work I have not only had to draw on my memory for much information derived from the works of others, but in many cases to consult those at the time. It could hardly serve any useful purpose were I to attempt a detailed enumeration of these. But a list is appended of those works from which I have most largely borrowed, viz.—Ophthalmic Hospital Reports; Von Graefe's writings; Helmholtz's Physiological Optics; Leber on the Retina and Optic Nerve;

Transactions of the Ophthalmological Society; Sattler on Pulsating Tumours of the Orbit and Graves' Disease; Fuchs on Uveal Sarcoma; Works on Glaucoma by Arlt, Schnabel, and Priestley Smith; Articles on Strabismus by Alf. Graefe and Hansen Grut; Berlin on Diseases of the Orbit; Papers on Lachrymal Affections and Inflammation of the Cornea by Hansen Grut.

31 Drumsheugh Gardens,
Edinburgh, *May* 1889.

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DISEASES OF THE EYE.

DISEASES OF THE EYE.

CHAPTER I.

EXAMINATION OF THE EYE.

The systematic examination of the eye must be both subjective and objective. In the *subjective examination* we have to question the patient as to any abnormal sensations which he may experience. We have also to determine by suitable tests how far the performance of the different visual functions, monocular as well as binocular, is in accordance with the normal standard. By the *objective examination* a more complete explanation may be found of the cause of any abnormalities which are revealed by subjective tests.

Considerable experience is sometimes required to know how far the result of subjective examination is in accordance with, and can be explained by, what may be found objectively. For instance, the question often arises—Does a certain degree of blindness correspond to certain opacities in the cornea or lens? Again, there may be very marked ophthalmoscopic changes producing little or no visual effect, whilst very serious defects of vision are sometimes due to changes which might readily escape observation.

Both the objective and subjective examinations of the eye should be made in a routine manner. This can be adhered to after a little practice without any sacrifice of time, and prevents any existing defects or abnormalities from being overlooked. On the whole it is advisable to complete the subjective before proceeding to the objective examination. But there are certain preliminary points in an objective examination to which attention should first be given. For instance, an inspection may be rapidly made of the lids, conjunctiva, cornea, and iris, and the intraocular tension, state of muscular equilibrium and power of convergence determined, before the subjective examination is begun. After the visual acuity of each eye has been noted, and any other subjective test which may be considered necessary applied, the objective examination may be continued in the dark room, first by examining the cornea, iris, and anterior surface of the lens by oblique illumination, then by reflecting light with the ophthalmoscope into the eye to ascertain the transparency of the other dioptric media, and finally by obtaining an ophthalmoscopic image of the different parts of the back of the eye.

PRELIMINARY OBJECTIVE EXAMINATION.

By *inspection of the lids* we note any abnormality of the skin and of the cilia, also whether the puncta lachrymalia are rightly applied to the eye, or at all everted. The position of the lids with respect to the eyes should be noted. By causing the patient to move the eyes upwards and downwards any abnormality in the lid movements may be detected. By placing the finger or thumb on the skin of the lower lid and slightly pulling it down, while the patient is directed to look up, the lower lid is everted, and the conjunctival surface brought into view. If this be normal in appearance it is unnecessary to examine the inner surface of the upper lid, but should the lower be injected or inflamed, or should there be any affection of the cornea or history of any foreign body having struck the eye, the upper lid must also be everted. This is done by raising the lid with the thumb of the one hand applied to its outer surface, so as to cause the eyelashes to project forwards. The eyelashes are then grasped between the forefinger and thumb of the other hand, and the lid in this way pulled downwards and forwards, while the patient is told to look down. At the same time the skin below the first thumb, which is slightly raised from its former position, is pressed downwards. Finally, a movement of rotation upwards of the other holding the margins of the lid, is made round it as a fulcrum. This causes the conjunctival surface to spring into view, and the more completely the more the eye is directed downwards.

Attention should also be paid to the condition of the *tear sac,* over which pressure may be made in order to see whether or not any regurgitation takes place. The importance of this is explained in the chapter on diseases of the lachrymal apparatus.

Inspection of the cornea may be rendered difficult by the more or less convulsive closure of the lids. This is of itself an indication that there is some irritation or inflammation of the cornea. Under such circumstances the eye should be opened by separating the lids with the finger tips applied to the lid margins. Attempts to separate the lids with the fingers otherwise than by securing the margins may be either altogether unsuccessful, and merely result in their eversion, or may not sufficiently expose the cornea. Desmarres' elevators (Fig. 1) should be used when there is a danger of bursting an ulcerated cornea. One elevator is inserted carefully under each lid and the eye opened by drawing them apart, avoiding at the same time any pressure on the cornea.

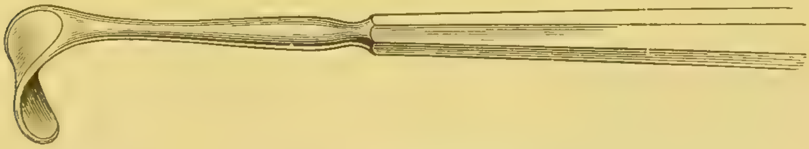


FIG. 1.—Desmarres' Retractor.

When the patient is placed facing a window any irregularity of the cornea is readily seen by the distorted image of the window reflected from the defective portion of the corneal surface. To observe this the patient is made to follow the examiner's finger as it is moved in different directions.

The *intraocular tension* may be tested by gently pressing the tips of the two forefingers on the eye through the upper lid, while the patient looks down. By alternately varying the degree of pressure of first the one and then the other on the eye, some idea is obtained of the resistance which it offers. Another and often more satisfactory way is to cause the patient to look up, and apply the tip of the forefinger of one hand directly to the conjunctiva of the globe below the cornea, making a slight tapping movement without altogether removing the finger from the eye. The interpretation of the impressions thus conveyed to the finger is referred to in the chapter on glaucoma.

The condition of *dynamic equilibrium of the external muscles* of the eye is determined, as far as is necessary for practical purposes, by causing the patient to fix a distant object alternately with either eye, whilst the observer's hand is held in front of the other, and by noticing whether any movement takes place in the eye which has been occluded on removal of the hand. If the eye be then moved inwards, there must have been a divergence; if outwards, a convergence of the axes when it was excluded from fixation. A more delicate and at the same time easily applied test is with a glass rod (Maddox's test). This test, the different conditions met with, and their bearing on the pathology of the ocular muscles, are discussed in Chapter XVII.

The *degree of convergence* is tested by causing the patient to fix a small object which is gradually approached to his face, and then noticing the shortest distance up to which the two axes continue to be directed towards it.

SUBJECTIVE EXAMINATIONS.

Visual acuity.—One of the most important points to determine in all cases, and one which, whatever be the complaint of the patient, should be tested as a matter of routine, is the degree of his visual acuity. This should be tested for each eye separately. The visual acuity is inversely proportional to the smallest visual angle under which two objects still give rise to distinct separate impressions. As the degree of absolute illumination is of influence on the acuity of vision, the ordinary test for the form sense should be made under a good illumination, such as is afforded by ordinary daylight or the light from a good gas burner placed close to, and reflected on to, the object serving as a test.

It has been found by experiment that two black objects on a white ground, if properly illuminated, can be seen by the normal eye as discrete, when separated by a space which subtends an angle of 1′ with the eye. That is to say, if the visual angle (in the figure, angle A or B) is equal to 1′. This limit, though not in all cases the very lowest, is found to be sufficiently low to form the basis for a practical scale of visual acuities. When the eye is capable of this degree of discriminating power, it is considered to have full visual acuity. If the smallest angle be, on the contrary, only 2′, 3′, 4′, the visual acuity is correspondingly expressed by the fractions $\frac{1}{2}$, $\frac{1}{3}$, and $\frac{1}{4}$.

Various test objects, prepared according to the principle just explained, are in use for the practical determination of the acuteness of vision. That most commonly used, and which has been found to be sufficiently accurate for practical purposes, is the set of test types introduced by Snellen, and generally known as *Snellen's test types.* These consist of a number of letters of the alphabet, or other figures, painted black on a white background. They are so formed that their height subtends an angle of 5′, while the spaces separating those portions which must be seen as discrete parts in order that the shape of the figure may be recognised, subtend angles of 1′ at the distances at which they should be read by any one possessing normal visual acuity.

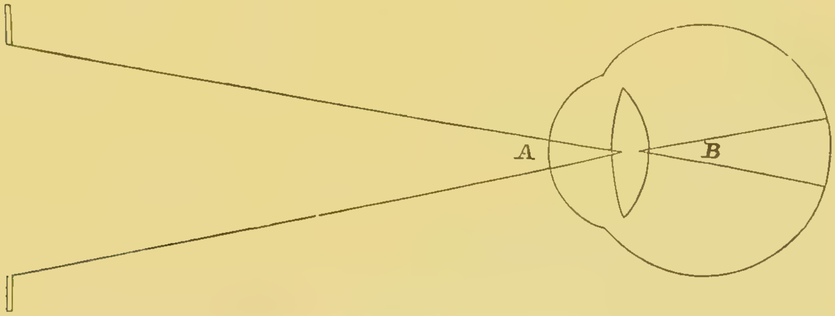


FIG. 2.—Diagram of visual angle.

A series of such letters or figures arranged as lines, the letters in each line being the same size, while there is a difference in the size of those in consecutive lines, constitute the set of test types. Above each line is placed a number, which indicates in feet or metres the distance at which the letters should be read. The number of lines usually employed is seven. Of these the one containing the smallest letters ought to be recognised at 20 feet, and the others should with the same visual acuity be recognisable at 30, 40, 50, 70, 100, and 200 feet respectively. Other tables have the distances marked in metres, the smallest letters being recognisable at 6 metres, and the others at 9, 12, 18, 24, 36, and 60 metres respectively. The smallest letters are taken, therefore, of such a size that it is possible to place a patient at a distance from them which can be conveniently obtained in most consulting rooms. Where the room is considerably less than 20 feet, or 6 metres in length, an additional line containing letters recognisable at 15 feet may be added.

With the aid of Snellen's test types the degree of visual acuity can, with sufficient accuracy, be expressed as a fraction, the denominator of which is the number giving either in feet or metres the distance at which the smallest type legible by the patient ought to be read, and the numerator that which on the same scale indicates the distance at which the individual tested is placed from the test. Thus, if at 20 feet distance the line marked 70, and which should therefore be recognised at 70 feet, can only just be made out, the acuity of vision is $\frac{20}{70}$, or, as it is often written, $V = \frac{20}{70}$. If the patient has to approach 3 feet nearer before being able to read that same line, $V = \frac{17}{70}$, and so on. When none of the types are seen at 20 feet,—that is, when (as the largest should be seen at 200 feet) the vision is less than $\frac{20}{200}$, the fraction expressing it may be found by approaching nearer and nearer to the types. If for instance, the largest type, and that alone, were first recognisable at 5 feet, the acuity of vision in such a case would be represented by $\frac{5}{200}$. In cases where the vision is very bad, it is customary to note the distance at which the outstretched fingers can be counted against the dark background of the examiner's coat, the result being noted as follows : V = fingers at 3 feet, or at 8 feet, as the case may be. When the vision is so far reduced that fingers cannot be counted even when close up to the eye, but yet the movements of the hand perceived, V is said to equal movements of the hand. This is often denoted thus : V = hand-reflex. When even this degree of acuity is not present there may still be perception of light, or V = perception of light (or V = P.L.).

The acuity, as tested by Snellen's test types, may not in all cases lead to expressions which are very strictly comparable, and, therefore, for purposes of scientific investigation, where great accuracy is aimed at, other test objects may be used. One of the best of these are Burchardt's so-called international tests, composed of a number of differently arranged dots, which should be counted at different distances. The inaccuracy connected with the ordinary Snellen's types lies mainly in the possibility of guessing what they are before they are very distinctly seen, a power which the educated possess in a higher degree than the illiterate, so that two individuals, with really the same acuity, might present apparent differences when the test is made with letters of the alphabet or other familiar figures. The main object, however, in making the test is to ascertain at different times, either in the course of any disease, or with and without optical correction, the degree of acuteness of vision in any particular case. This is for practical purposes sufficiently, accurately, and most rapidly determined by Snellen's test types, which have attained a popularity very much greater than other similar, and in most cases less accurate, tests.

Besides the acuity of vision, or the acuity of the sense of form, two other functions of the central portion of the retina sometimes call for investigation, viz., the colour vision and the sense of light, independently of form and colour.

The method of testing the *central colour vision* is fully discussed under colour blindness in Chapter XIV.

Light Sense.—When an examination of the *light sense* is made, it is necessary to gain information with respect to two different points: (1.) The amount of illumination which is just sufficient to give rise to a sensation of light; and (2.) the smallest difference between two intensities of illumination which is capable of being distinguished. It is difficult to make the examination in such a way as to clearly discriminate between pure sensations of light and mixed sensations of light and form, as they all but invariably present themselves to our consciousness. But, in the examination of the light sense as in all other subjective examinations, whatever their nature, our object is not to get mathematically accurate results, as that is impossible, but only such for which the limits of error are not too wide for practical purposes.

The first element in the light sense is most conveniently tested by means of Foerster's photometer. With it the degrees of absolute illumination required to render a particular object only just visible is measured. The result thus obtained, when compared with the normal standard under similar conditions, enables us to express the acuity of the light sense in the form of a fraction.

Foerster's photometer consists of a rectangular box one foot long and rather less than a foot in breadth and height, blackened inside, and fixed to an adjustable stand. To one side of one of the ends of this box, two apertures, prolonged outwards into short projecting tubes, are placed for the eyes to look through. At the side of these apertures is the arrangement for illuminating the interior of the box. This consists of a standard candle kept always at the same height by means of a spring, and placed at a short distance from the hole or window in the box. The size of this window (which is covered with white paper), and consequently the amount of light which enters the box, is regulated by an adjustable rectangular diaphragm, the centre of which always remains in the same position. By knowing the area of this diaphragm it is easy to calculate the corresponding degrees of illumination. At the other end of the box is placed the object to be recognised,—most conveniently a couple of squares of white paper about one inch apart, and also about one inch in size.

A difficulty in connection with all methods for determining the minimum perceptible quantity of light arises from the enormous degree to which that quantity varies, according to what is known as the state of adaptation of the retina. The sensitiveness to light is many hundred times less when the eye has been exposed for some time to strong daylight, than it is when it has been for some time entirely kept in the dark. Practically, then, the unit must be taken from a comparison with one's own light perception under the same conditions, and with proper care that the patient examined understands the nature of the test which is being made.

The second element in the light sense, the power of discriminating between impressions of different intensity, may be tested by means of what is called Masson's disc.

This consists of a disc of white cardboard fixed at its centre to an apparatus of multiplying wheels, by which it can be rapidly rotated on its axis. On the white surface are painted a number of black marks, which are of such a shape as to form portions of sectors of the disc of different sizes. These black sectors produce, on rapid rotation of the disc, a number of grey rings, the intensity of which varies. Thus the difference of intensity between the light reflected from the white surface alone, and the grey rings produced by the combination of the blending of the impressions from it, and a black sector of 4°, is practically $^1/_{90}$; with a sector of 6° it is $^1/_{60}$, and so on, the difference of intensity in background and ring, or

$$\mathrm{I} - \mathrm{I}' = \frac{x^\circ}{360^\circ}.$$

Although the normal power of distinguishing between degrees of intensity varies with the absolute illumination, the amount of such variation is slight, and does not cause any difficulty in the examination, the result of which can always be controlled by a comparison with what one sees one's-self. The rings should be of considerable breadth, not less than half-inch, so that their visibility at the distance of one foot may be independent of the acuity of vision in any case where the test is at all applicable. If under any particular illumination the examiner, who has no defect of the light sense, can just see the ring formed by the sector of $x°$, while the last seen by the individual examined is $y°$, the fraction representing the acuity of this element of light sense may be, for purposes of comparison, conveniently taken as $\frac{x}{y}$.

If we leave out of consideration the question of the light sense for the peripheral portions of the field of vision, the accurate examination of which is in any case a matter of some difficulty, there seems no reason why any attempt should be made to altogether dissociate the light and form senses. It is sufficient for practical purposes, as was first pointed out by Bjerrum, to recognise the abnormal manners in which the form sense is influenced by the conditions of illumination. It is a matter of experience that where the minimum perceptible degree of illumination is very appreciably greater than normal, a marked diminution in the visual acuity is found under an illumination

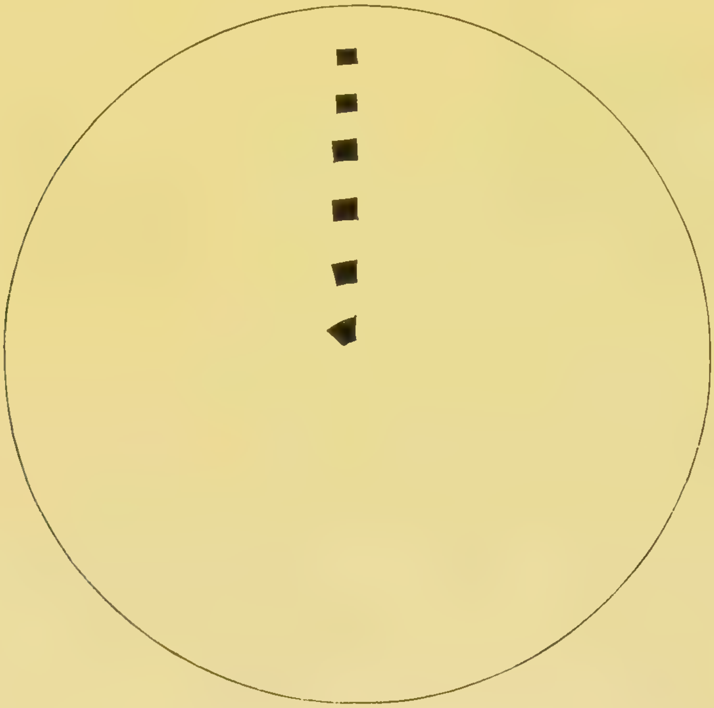


FIG. 3.—Masson's Disc (one-fourth diameter).

which either does not at all affect, or it may be only slightly affects, the acuity of the normal eye. It is evident, too, that an eye which is very deficient in the appreciation of differences of intensity of illumination will be influenced in its visual acuity for objects the separate and distinguishable parts of which are not strongly contrasted in their relative luminosity. One of the simplest practical methods, then, of testing the first element of the light sense is to diminish the illumination until it just begins to affect the acuity of one's own (presumably normal) eye. An equally practical test for the second element exists in Bjerrum's test types, which consist of Snellen's types printed grey on grey. The contrast between the letters and the background is much less than for the ordinary types, which are printed black on white.

For the determination of the acuity of the light sense in cases of cataract, a form of photometer employed by von Graefe is useful in doubtful cases. This photometer consists merely of a darkened box, in the inside of which is a candle, and on one side of which is a window of milk glass, the size of which can be regulated at will.

The vision of the peripheral portions of the retina may be defective with respect to all or any of the senses of form, colour, and light. As the nature of such defects often throws light on the diagnosis and prognosis of a case, the testing of the peripheral retinal functions takes an important place in the subjective examination of the eye.

Peripheral vision may be defective in continuity or in acuity: often there is a defect in both respects at the same time, that is to say, that the most peripheral portions, where the normal acuity is least, may have their acuity reduced to 0, while the same course of impairment reduces, but does not altogether abolish, the acuity of the more central portions. But besides regular interruptions in the continuity of the field of vision, which manifest themselves by some limitation, in the normal extent of the field in some particular direction, or in all directions, irregular breaches in continuity, or more or less blind portions surrounded by normal or relatively normal portions of the field, are met with, to which the name of *scotomata* has been given.

To make an accurate examination of the field of vision it is necessary to make use of some sort of perimeter. Such an examination takes some little time, and is only called for in certain cases. A rough examination, which is sufficient for the determination of any existing limitation in the peripheral boundaries of the field, should be made as much a matter of routine as the taking of the central visual acuity. It may be made in the following manner. The patient is placed with his back to the light, and facing the observer. He is directed to cover one eye with his hand, and to fix steadily with the other the eye of the observer, which is directly opposite his own; therefore, if the left be the one to be examined, the patient fixes with his left eye the right eye of the observer, whilst the observer at the same time fixes with his right eye the patient's left. In this position there is obviously a plane at right angles to the common line of fixation, and cutting that line at its mid-point, any point on which, provided rays from it enter the two eyes, will have an image at correspondingly situated points on the two retinæ. If, therefore, the observer, on moving his hand in any direction in this plane from or towards the middle of the line of fixation, finds that it disappears from or comes into his

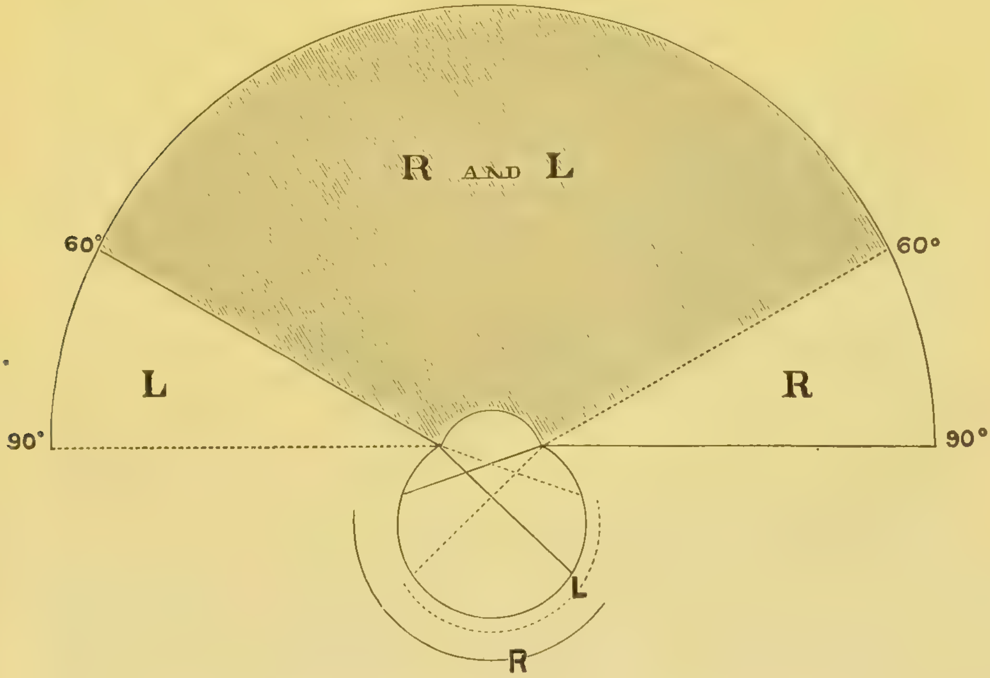


Fig. 4.—Diagram of Field of Vision.

own and the patient's view simultaneously, it follows that their fields of vision are co-extensive in that direction. A test in this manner can be rapidly made in all directions, and a want of coincidence of the two fields in any or all directions be easily discovered. This method of testing is only, however, a qualitative one, in so far as by it the existence and not the extent of a limitation is determined.

For the purpose of obtaining more accurate measurements of the position and extent of any deviations from the normal character of the field of vision, as well as of estimating the peripheral visual acuity, a perimeter is required. The result of a perimeter examination, too, is most conveniently recorded on a chart, which consists of a number of concentric circles, the radii of which increase by an equal amount, and a number of radiating lines from their common centre. The circles, of which there are usually nine, represent at intervals of 10° the angular aperture of the field; the lines the different meridians. In the charts commonly used the centre of the figure, therefore, corresponds to the point of fixation, whilst the concentric circles represent the portions of the field whose retinal images are equidistant in all directions from the fovea. If there be any doubt as to the restriction of the field in any particular case, the examination is best made in a subdued light, the intensity of which is a little greater than that which begins to tell on the normal field. This is a sort of compromise between the examination of the light and form senses, which is of considerable practical importance.

The normal extent of the field of vision is subject to variation at the upper and also (though to a less extent) at the inner parts, owing to individual peculiarities in the size and shape of the eyebrows and nose. The physiological limits may be taken to be as follows:—Upwards, 45°; upwards and outwards, 50° to 55°; outwards, 90° (often slightly more); outwards and downwards, 80° to 85°; downwards, 70°; downwards and inwards, 60° (variable on account of the nose); inwards, 55° to 60°; inwards and upwards, 55°. The extent of the field upwards, and upwards and inwards, is found to be 5° to 15° greater when the point of fixation is situated 20° or 30° from the centre of the perimeter in the opposite direction. This has to be borne in mind when there is a doubt as to whether or not there is limitation in this region. Thus, should the exploration with the centre of the perimeter as point of fixation only give an angular dimension of 40° for the peripheral extension of the field upwards, we should not always be right in assuming a contraction in this direction, unless no increased measurement resulted from testing with a lower point of fixation.

The simplest form of perimeter consists of a black board and a semicircular arm of blackened metal, of one foot radius, from the middle point of the convex surface of which there is a projection which fits into a hole in the board, and permits of the arm being rotated round its own diameter, that is, round a line at right angles to the board. Radiating lines are scratched or cut on the board from the central hole, and separated by definite angular distances (30° or 45°). These indicate the position in which the semicircular arm is to be placed for any particular meridian in which the extent of the field has to be tested. The arm itself is graduated in degrees by lines 5° apart, and starting from 0° at the centre to 90° at either end. The patient is seated in front of the perimeter, with his chin resting on a support so arranged that the eye to be examined comes to occupy a position at the centre of the semicircular arm, or as nearly as possible one foot from the point of fixation. The observer causes a white object—*e.g.* a square of white paper at the end of a small black rod — to travel along the arm, and notes when, whilst the patient fixes the middle of the arm, it ceases to be seen if moved from the centre outwards, or first comes into view if moved in the opposite direction. The two points thus found to either side of the centre in each meridian are then noted on the chart, or recorded in any other manner which may be found convenient. Thus, for instance, the extent of the field in eight directions along meridians separated from each other by angles of 45° may, as Priestley Smith has suggested, be conveniently noted in the following manner, which represents the normal field:—

55	45	50	50	45	55
90	R	60	60	L	90
85	70	60	60	70	85

The white test object should not, as a rule, exceed 10 millimetres square, and the observer should stand in such a position as to make sure, by watching the patient's eye, that he constantly fixes the centre of the perimeter.

It is a good plan to have a few concentric rings cut on the board itself, the radii of which correspond to the projections on it of the scale on the arm. Small scotomata (either involving or situated near the point of fixation), or the limits of very constricted fields, can then be accurately marked out on the board.

The maximum extent of the field of vision is only got for objects seen under a visual angle of $\frac{1}{2}$° and more. For smaller visual angles there is a gradual diminution in the size of the field. The test objects generally employed in perimetric measurements are from 1 to 2 centimetres in diameter, which, at a distance of 30 centimetres, or about the radius of the perimeter arc, correspond to a visual angle of from 2° to 4°. Their images must, therefore, cover many thousands of retinal elements, so that the test is far from being a fine one, and not at all comparable to the tests made for direct vision. A useful addition to the usual method of examining the field of vision consists in making use of white objects which subtend a very small visual angle. In order, however, to be able to work without difficulty with sufficiently small visual angles, it is necessary to have the patient placed some distance from the plane in which the test objects lie. A method of examination based on this principle was introduced not long ago by Bjerrum, and is capable, in many cases, of affording data of considerable diagnostic importance. A large black screen, 2 metres broad, which can be let down from the ceiling to the floor, should be used—the screen is most conveniently placed on the wall opposite the space between the windows, so as to get good light all round. It has to be pretty large, as the projection of the blind spot at a distance of a couple of metres, instead of measuring about 1 inch, as on an ordinary perimeter, where the distance of projection is 1 foot, or 30 centimetres, measures 7 inches in diameter, and everything else is, of course, in the same proportion. A screen, 2 metres broad, will admit of testing up to about 27° from the point of fixation, if that be in the middle of the screen, while, by removing the point of fixation to the edge of the screen, a larger field can be measured. When the test object is small, the central point of the screen can be used, and this, of course, is the most convenient arrangement. The test objects used by Bjerrum are small circular discs of ivory fixed on the end of a long dull black metal rod. They are of different sizes, from 10 millimetres to 1 millimetre in diameter. The examination is begun with a disc of 10 millimetres diameter, at the ordinary distance (30 centimetres), and afterwards continued in suitable cases with one 3 millimetres in diameter, at the distance of 2 metres. In the first case the visual angle is $\frac{10}{300}$, in the second $\frac{3}{2000}$, or approximately 2° and 5′ respectively. In the case of the 5′ visual angle the boundaries of the normal field, instead of being as extensive as they are found to be by the ordinary method of examination (see p. 13), average 35° outwards, 30° inwards, 28° downwards, and 25° upwards. Too much weight must not be attached to what appears to be slight concentric limitations got from an examination by the small visual angle method. It is the irregular limitations, the more or less sector-shaped defects and blind areas (scotomata), which are of the greatest importance. Concentric limitations are met with by this test as individual peculiarities. Under normal conditions, however, there are never found to be marked indentations or scotomata in the diminished field. Variations in illumination, it must be remembered, too, have a somewhat greater influence on the results got by testing with the small images than in the case of the ordinary perimetric method.

Sometimes it is useful to test not only the continuity but also the *visual acuity of different parts of the field of vision.* If an ordinary perimeter be used, the test object for this purpose is most conveniently a figure composed of two black squares separated from each other by their own length. With the object of controlling the statements of the patients as to whether or not at any particular part of the field these two squares are seen as distinct objects, a black rectangular figure of the same breadth, but three times the length of the squares, may

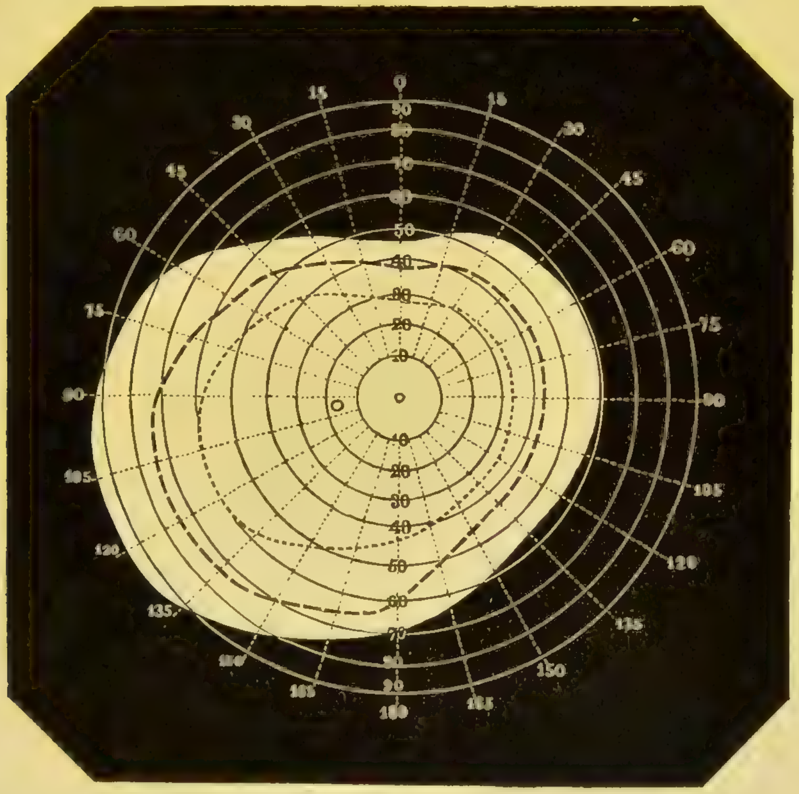


FIG. 5.—Perimeter chart of field of vision; — for white, ... for a red and complementary green, - - - for a blue and complementary yellow.

be alternately placed in the same position. This may readily be done by having the two figures on either side of a piece of paper or cardboard. With such test objects of different sizes, the visual acuity for any part of the field can be tested and compared with that of the same portion of the field of the observer. Or, as is occasionally useful, for instance in cases of incomplete hemianopia, the distance from the centre at which a definitely sized test object of this kind can be just made out is compared with the distance at which it is seen on the same side of the other eye, or the opposite side of the first eye. The most trustworthy test for peripheral visual acuity, however, is that made with small objects at a distance, as in Bjerrum's screen test just described.

The determination of the *limits of the field for different colours* may be made with pieces of coloured paper. The results thus obtained are influenced by the hue and shade of the colour used, by the size of the coloured objects, and by the quality and intensity of the light under which the examination is conducted. Practically, in examining any case it is useful to determine the extent of one's own visual field in one or two directions under the same conditions, and then allow something for individual peculiarity besides. Another precaution that will be found useful is to have the test object (which it is well to make 20 millimetres square) differently coloured on either side. In this way we are provided with a check on the accuracy of the patient's statements. The most convenient colour to use is some hue of red, as it is for the reds and greens that pathological defects in the colour sense first manifest themselves. If the test colour chosen be examined carefully at the inner side of the field of vision, it will generally be found to change colour. Before becoming absolutely colourless, it will become yellowish, brownish, or bluish, according to the hue and shade selected, and it is this tint which may with advantage be selected for colouring the opposite side of the test object. When the red-green perception is entirely abolished, the limits may be taken for blue or yellow, the vision for which is almost always longer in disappearing; but the only necessity for this is to exclude the possibility of the more usual form of congenital colour blindness, which we should have reason to suspect if the peripheral boundaries for yellow and blue were not restricted.

What are called self-registering perimeters are now much used, and render the examinations of peripheral visions more easy and rapid. A number of different perimeters of this kind, with which the chart representing the size of the field of vision can be more or less automatically obtained at the time of the examination, are in use. Of these the perimeters of Priestley Smith, M'Hardy, and Blix, are perhaps the best.

SUBJECTIVE SENSATIONS.

The different subjective sensations which may be complained of, and for which a proper objective examination will afford a more or less satisfactory explanation in most cases, need only be shortly referred to in this chapter, as they are mentioned in connection with the diseases in which they are mostly manifested. The principal ones are pain, double vision, distorted vision, the appearance of spots or clouds in front of the eye, and subjective light and colour sensations.

Pain.—Pain complained of in the eye may be non-inflammatory. It may also be primarily due to inflammation of the eye or the surrounding parts.

In a large proportion of the cases of pain unaccompanied by inflammation, the pain only comes on, or is at all severe, when the eyes are used for work near at hand, such as reading or sewing. In other cases, again, the pain is independent of the use of the eyes. The name which is generally given to the non-inflammatory pain associated with the use of the eyes is *asthenopia*, which literally means a want of power in the eye to perform its functions. As a general rule, the greater the pain complained of, the less likely is it to depend upon any pathological condition at all, which can be referred to the eye. But it is necessary to make a thorough examination into the state of refraction and the condition of the external muscles, before it is possible to exclude those forms of asthenopia which depend on accommodative or muscular difficulties, the symptoms of which are elsewhere discussed.

Pain in the eyes, often described as at the back of the eyes, unconnected with any accommodative or muscular abnormality, and coming on, often very severely, at longer or shorter intervals after use of the eyes for reading, &c., is extremely common. Often the condition is associated with more or less sensitiveness to light. In many, and indeed most of these cases, there is absolutely nothing to be found, in connection with the eyes at any rate, to account for the persistence or severity of the symptoms. Either the strain on the attention becomes soon too overpowering, or the retina itself is hypersensitive and easily tired; at all events, there is some weakness in the tone or capabilities of the nervous mechanism of vision. Such a state of *nervous asthenopia* is probably frequently of reflex origin, though the region from which the afferent stimulus proceeds is not often apparent. It is more common in women than in men, which has led some authors to ascribe it to uterine irritation, and even to describe a particular form of chronic inflammation with which it is associated. The causal connection between the two, as a matter of constant or even frequent occurrence, is, however, in the highest degree improbable. Intestinal irritation also appears sometimes to give rise to this form of asthenopia. In a great number of cases—and I am disposed to think in by far the greatest number—the pain is due primarily to over-exertion of the eyes at a time when, after a debilitating illness, or a state of malnutrition, anæmia, &c., the nervous tone is below par. The pain thus originating becomes chronic, a sort of neurosis or habit, and may become so distressing as to render reading impossible for more than a few minutes at a time. Sometimes the state of the eyes themselves is such as to induce asthenopia from over use. In congenital amblyopia and astigmatism, for instance, where the difficulties in deciphering small type are considerable, reading may cause an abnormal strain. Indeed one can bring on the same kind of feeling in one's own eyes by attempting to read small type either beyond the limit of distinct vision, or with an illumination barely sufficiently powerful for that purpose, and independently of over-exertion of convergence or accommodation. Individuals suffering from this form of asthenopia generally take sooner or later to the use of blue or dark spectacles, and by doing so often merely increase tbe sensitiveness of their retinæ. Such spectacles should only be employed in cases of deep-seated inflammation, in which the tempering of the light passing into the eyes is one important means of complying with the indication for rest, or under conditions of excessive glare from powerful sources of illumination or reflection.

Many cases of retinal or nervous asthenopia are amongst the most severe and rebellious that one is called upon to treat. Cold water douches, and iron internally, are indicated in some cases; others are much benefited by hot sponging or fomentations; and where there is absolutely nothing abnormal to be discovered in the eyes, an energetic attempt should be made to break the habit of giving up reading as soon as the pain comes on. This can only be done gradually, but is successful in many cases.

Pain, independent of the use of the eyes, is often complained of, and is generally of a neuralgic character, and more or less intense. The diagnosis is easily made when there are points of special tenderness around the eye. Occasionally, no doubt, the pain is reflex, but the origin of most cases is a hypersensitiveness of the supra- and infra-orbital branches of the fifth nerve, generally caused originally by exposure to cold.

When the pain is due to inflammation the diagnosis is often aided by noting the circumstances under which it is most intense. Thus sometimes it comes on mostly at night, at other times mostly when light falls into the eye, or when the eye is moved in some particular direction, or by pressure on some parts of the eye itself, or of the surrounding structures.

Diplopia.—When double vision is complained of, we have, in the first place, to determine by covering first the one eye, and then the other, whether in either case it is still present, or whether it always disappears when one eye alone is used; that is to say, whether the diplopia is monocular or binocular. If there be a fair amount of vision in both eyes, and no abnormal degree of suppression by the mind of the image falling on the misdirected eye, there will usually be binocular diplopia when both axes are not simultaneously directed on the same point of the object which engages the attention. There may be no diplopia complained of even though there be wide divergence of the visual image of the axes, owing to the unconscious mental suppression of the image of the one eye when the other is used for fixation. This suppression is sometimes so complete, that under no circumstances can the diplopia be made apparent. In other cases, by holding a red glass or a prism with the angle directed upwards or downwards in front of the fixing eye, the faulty image of the other is at once seen, showing that the suppression is only effected for normal conditions of similarity in the optical images and only for a retinal area, on which, under ordinary circumstances, the images corresponding to those occupying the centre of the retina of the fixing eye are received.

Again, an individual may complain of diplopia even although the visual axes are capable of crossing, and actually do cross, on the same object. This is the case when other objects than the one fixed engage the attention, and is, in fact, physiological, in so far as most objects not directly looked at are seen double. When, therefore, this kind of double vision is complained of, it is owing to an abnormal degree of attention being directed to objects other than those fixed. Usually there is a suppression of one of the images of other objects than the one on which the visual axes are directed—a suppression which is often so complete as to render it difficult for many people to become conscious of this physiological diplopia. Which eye is the one whose images are suppressed in any particular case may be determined by a very simple experiment. By asking any one, while keeping both eyes open, to hold up their finger in a line with some distant object, and then close first the one and then the other eye, they will generally find that the finger exactly covers the object as seen by one eye, while it deviates to one side when looked at with the other. This shows that only the image formed in the eye, in a line with which and the distant object the finger has been placed, is observed, that in the other being more or less completely suppressed. The cases of diplopia just referred to are due to the opposite condition, viz., to a too ready appreciation of the physiological double images. This is mostly met with in women, but occasionally also in men. When once discovered by them, and not recognised as physiological, they contract a habit of directing attention to the double images which sometimes becomes almost painful. Occasionally it is the nose which is in this way brought prominently before their notice, and which always appears to get in the way of other objects, by attracting attention at the same time; at other times, any objects, both beyond and nearer than the point of fixation, are continually forcing their two images on the attention.

Patients who suffer from true diplopia, due to a misdirection of one of the visual axes on the objects fixed, generally complain most of the doubling of that object, one image of which they usually recognise as the "true" image, that is, belonging to the properly directed eye, and the other as the "false," the image of the misdirected eye. The false image not only occupies a distinctly abnormal position, but is besides more or less indistinct, owing to its corresponding to a peripheral and not a central impression. Although, however, we can infer from observing to which eye the true image belongs, which is the fixing eye, it does not by any means follow that the defect in muscular activity is to be found in the other, as the cause of diplopia may be a condition common to both eyes, or confined to the fixing eye alone, the eye made use of for fixation being often determined by causes altogether unconnected with the condition of which the diplopia is a manifestation. Occasionally, whilst recognising the doubling of the object fixed, the more distressing symptom complained of is a veiling of that object by the projection over it of the image falling on the fovea of the misdirected eye, so that two different objects are constantly seen, the one through the other, as it were, just as the two images of a stereoscopic picture are projected to the same position in space.

The conditions under which the diplopia manifests itself have to be inquired into and tested. Thus, in many cases where it is complained of, it is not present under all circumstances. This may be due to want of constancy in the conditions giving rise to it, as is the case in periodic squint, or to the fact that the double vision only exists for certain directions of fixation. Again, in cases where there is a defect of convergent power, or an increased tendency to convergence, which may even amount to spasm, there is often diplopia within or beyond a certain distance from the eye—a distance which not only varies according to the degree of the abnormality, but also according to the inclination of the visual axes above or below the horizontal plane of fixation.

The manner of testing for diplopia, as well as the interpretation of the results thus obtained, is fully discussed in the chapter on muscular anomalies.

When the diplopia complained of is found, in the way already described above, to be monocular, the cause will generally be some error of refraction which has long existed or has been lately acquired, and a careful examination will be necessary to discover it. We have first to find out whether the diplopia has suddenly made its appearance or not. If so, it may either be due to some trauma, or the conditions giving rise to it may have previously existed, and only be observed owing to some circumstance which has caused the patient's attention to be directed to it. If there has been a trauma, we may look for irregular astigmatism, or some refractive changes, due to alteration in the position or condition of the lens, &c. The most common causes of acquired monocular diplopia are changes sometimes occurring in the lens during the formation of cataract, and alteration of the curvature of the cornea produced by inflammatory changes.

The defect of spherical aberration, common to all refracting surfaces which are not of such a form as to neutralise it, and therefore not what is called aplanatic, and which in optical instruments is practically to a great extent overcome by a centered system of different curvatures and different intervening distances, is to a great extent also rectified in the eye, partly owing to the dioptric arrangements, and partly no doubt also to the isolation of each percipient element of the retina. But there is a form of monochromatic aberration in the eye which does not exist in properly constructed optical instruments, which gives rise under certain conditions to polyopia. This aberration is due to the circumstance that the foci for rays passing through different portions (sectors) of the crystalline lens do not all coincide. When the eye is properly accommodated the want of coincidence of the foci is not sufficient to give rise to separate images, the only effect of the condition being that the retinal image of a point is larger than it would otherwise be. If, however, the retina be out of focus, the different images, instead of partially overlapping, are seen more or less distinctly as separate images. These images are crossed if the retina lies in front of the focal plane, and homonymous if it lies behind that plane—that is to say, on partially occluding the pupil, the images of the opposite or of the same side disappear respectively. This kind of polyopia is very evident in cases of faulty refraction (ametropia), when a bright object for which the eye cannot be accommodated is fixed, and it is an exaggeration of this phenomenon which is the cause of the polyopia occurring in commencing cataract. When not due to a trauma, the doubling of the image in one eye, hitherto existing, though unobserved, may sometimes suddenly force itself on the attention, either owing to increased retinal susceptibility to impressions, or to some cause having temporarily disabled the other eye; and when once discovered it may become more or less troublesome. In this way we sometimes find monocular existing along with binocular diplopia, the former being first brought to notice by the occurrence of the latter.

Metamorphopsia.—When distorted vision, or metamorphopsia, is complained of, it should suggest either some cause altering the character of the impressions formed on the retina, as, for instance, irregular astigmatism, or some pathological change which interferes with the normal manner in which our judgment of the shape of objects seen is formed. The apparent shape of objects depends, in the first place, on the configuration of their retinal images; it is therefore more in accordance with their actual shape the more regular is the refraction of the rays through the dioptric media of the eye, and the more accurately the rays are focussed on the retina. For the correct appreciation of form it is further necessary that the percipient elements of the retina should be grouped according to their normal arrangement in the focal surface of the eye, and should all be capable of excitation to some extent at least.

There is also, of course, the psychical element, which here, as always when there is a question of the interpretation of the impressions of the senses, has to be taken into account. Various *illusions* and *delusions* occur as to form and dimensions, as they do in connection with other impressions and other special senses. These constitute, however, for the most part at least, a chapter in the physiology and pathology of the nervous system, and do not call for consideration as symptoms of disorders of the eye.

Of the size of objects, again, we judge partly from the size of their retinal images, but as this, besides depending on the actual size of the objects, depends also on their distance from the eyes, the appreciation of size is more correct the more accurately the distance is gauged. In comparing the sizes of two or more objects, we are probably guided by the appreciation of their retinal images alone, if they are small. When of any considerable size, however, the method of comparison is more complicated, and depends chiefly upon the knowledge gained by the muscular sense on running the eyes along over the different points of the objects.

Defects of accommodation give rise to alterations in apparent size, owing to the error in the judgment of the distance of objects within the ordinary range of accommodation which results from them. Thus, where all at once there is a paresis of accommodation, and a greater effort has to be made in order to focus a near object distinctly, or to attempt to focus it distinctly, this gives rise to an impression of greater proximity of the object, and as the image on the retina is of course the same size as it would be were its distance more correctly estimated, it appears smaller. The micropsia thus occasioned is often very marked, and is met with both in cases where the paresis is the result of a direct, and where it is due to a reflex, interference with the functions of the branches of the third nerve which supply the ciliary muscle. The paresis may occur idiopathically, or be due to the action of a mydriatic. Conversely, a spasm of accommodation, from whatever cause, sometimes gives rise to macropsia, as the consciousness of an abnormally feeble effort to accommodate the eye for a near object causes the object to appear more distant, and therefore bigger than it would be otherwise estimated. Macropsia has been observed after the use of such myotics as pilocarpine and eserine. Accommodative micropsia is most marked the nearer the object lies to the eye: accommodative macropsia, on the other hand, the further it is removed from the eye.

But, besides metamorphopsia due to defects in accommodation, there are also similar misjudgments as to size, caused by abnormally impeded or facilitated movements of convergence of the optic axes, or by any optical conditions which permit of binocular fixation with the axes of vision directed so as to meet either nearer or further off than the object looked at. Thus, prisms with the bases inwards in front of each eye appear to magnify owing to the axes of vision meeting further off than the objects looked at. Prisms with the bases outwards in front of each eye appear, on the other hand, to diminish, as under these circumstances the axes of vision cross in front of the objects fixed, which appear nearer and consequently smaller than would be the case without the prisms. Operations on the muscles of the eye are sometimes, if there has been previously binocular vision, followed by similar apparent alteration in the size of near objects. Metamorphopsia due to alterations in the percipient elements of the retina, brought about by choroidal exudation, is described in the chapter on diseases of the choroid.

Scotomata and clouded vision.—When a patient complains of seeing a spot or spots in front of the eye, we have first to inquire whether the appearance is stationary and constant, or is only seen when the eye is suddenly moved, or has an independent motion. Its behaviour in these respects gives a clue where to look for the cause. When stationary and always projected in the same direction with reference to that taken up by the eye, it will either be due to some circumscribed opacity of the cornea or lens, possibly also of the vitreous, or to some change in the retina. An interruption in the field of vision of this last nature is called a positive scotoma. When there is no consciousness of an interruption corresponding to the more or less blind area, the scotoma is said to be negative.

Where a lesion primarily involves the nerve fibres of the retina the resulting scotoma is unperceived or negative; where, on the other hand, lesions involving the retinal pigment and choroid cause the scotoma, there is more or less consciousness of its existence. This Foerster explains by assuming that there is unequal stimulation of the light-perceiving elements in the defective and surrounding areas, the subjective sensation associated with any lesion giving rise to torpor of the retina being that of relative darkness. The shape of a positive scotoma may often be learnt by asking the patient to draw the outline of it on a piece of white paper. Positive scotomata are generally most marked in subdued light. Where the spot is not stationary it is generally due to the shadow cast by something in the vitreous. A less common and very apparent cause is the movement of a dislocated and opaque lens. Small spots, or *muscæ volitantes*, which cannot be detected with the ophthalmoscope, are very commonly complained of (see muscæ). *Clouded vision* is often met with, and should direct attention to the choroid or to the possible existence of glaucoma. If intermittent in its character, there will often be reason to suspect glaucoma. Patients suffering from glaucoma often complain of seeing everything clouded even when found on examination to have full visual acuity.

Sensations of light and colour.—These may arise even when there is no objective cause to account for them—that is, even when no undulations of the ether, such as are capable of giving rise to luminous impressions, find their way to the retina. A purely subjective sensation of this kind may be the result of a mechanical or chemical stimulation of the nerve fibres of the retina or optic nerve, or of the centres of vision. The light so seen may appear coloured or uncoloured, according to the site and nature of the stimulation. Further, under certain circumstances, coloured luminous impressions may be received when the objective source is of a nature which should only give rise normally to uncoloured light sensations. This may be due either to causes which separate the compound white light into its constituent rays, or to such as give rise to a selection of some rays, and at the same time a greater or less absorption of others.

Direct pressure over a portion of the eye behind its equator produces in the dark a so-called *phosphene* or sensation of light, which is referred to the opposite side of the field of vision. If a similar pressure be made in the light instead of in the dark, it is a darkish spot which is seen, the retina being apparently rendered by such mechanical irritation less responsive to stimulation by light. The slight stretching to which the retina is subjected by the action of the ciliary muscle also gives rise on its sudden discontinuance to a faint phosphene, which may be experienced by suddenly relaxing the accommodation in the dark. Phosphenes also occur in strained movements of the eye. These appear to be most marked on turning the eye upwards, and are probably due more to intermittent pressure of the oculo-motor muscles in the eyeball than to stretching of the optic nerve by the movement.

The presence or absence of colour in the phosphenes, due to stretching of the retina from any cause, may be of importance as affording some indication of the portion stretched, apart from that which is indicated by the locality of the field to which they are projected. The most peripheral portions of the retina are either totally colour blind, or require a very much greater stimulation to give rise to coloured impressions than more central portions. Consequently, when they alone are mechanically stimulated the phosphenes are colourless. As a general rule, indeed, colourless phosphenes are due to stretching of the anterior part of the retina. Such colourless and indefinite phosphenes are complained of, too, where there is slight cyclitis. They take the form of more or less incomplete circles, and, as they are not constant, are in all probability brought about by movements of the ciliary muscle, which cause dragging of, or pressure on, the hypersensitive anterior part of the retina.

A common cause of coloured vision, owing to a change produced on the compound rays of white light as they enter the eye, is afforded by those states of the cornea which give rise to diffraction. In looking at the street lamps, for instance, through the glass of a carriage window, which is covered with the closely packed particles of moisture which arise from the condensation on it of the vapour within, one frequently notices the lights surrounded by coloured haloes. The same appearance may sometimes be observed without the intervention of the glass, and it is then usually pathological and due to a similar arrangement of intransparent particles in the cornea. This symptom of seeing haloes round lights is one of the commonest and most important indications of the early stage of glaucoma. Although a frequent symptom in glaucoma, such coloured haloes are by no means pathognomonic of that disease. They may be seen in all cases where the cornea assumes from any cause a diffuse cloudiness. Even the secretion which in some cases of conjunctivitis gathers across the cornea may give rise to them. When spontaneously complained of, and especially when there is no appearance in the cornea at the time of examination to account for the halo, and when, too, it does not disappear on rubbing the eyes, its existence is strongly suggestive of glaucoma.

Test for the presence of binocular vision.—It is sometimes necessary to determine whether in any case binocular vision exists or not. This may be done with a stereoscope and suitably selected pictures, or by Hering's method.

I have found the following a convenient stereoscopic test to adopt. The test consists in altering simultaneously the pictures presented in the stereoscope to either eye, so that the impression given also varies. If, for instance, in Fig. 6, the small circles occupy the positions A and B respectively, the stereoscopic impression is that of a *hollow* cylinder or cone, with the small circle appearing further away than the large one. On the other hand, when the small circles come into the positions A′ and B′, the cone appears in relief and solid, the small circle appearing next the eyes. If all stages of the transition between these two positions be presented to the eyes (as is done by a simple mechanism causing a lateral approximation and separation of the small circles) the stereoscopic effect is one of movement in the third dimension. The small circle then appears to rise from a plane lower than the picture to one which lies nearer the eyes. This apparent movement is so evident, especially if the experiment be made in semi-darkness, that young children can at once say whether they can see it or not. Seeing it implies the existence of binocular vision.

In Hering's method the patient is caused to look with both eyes through a tube blackened inside, and across one end of which a string is stretched. If a small object, such as a bead or pea, be dropped immediately in front of or behind the thread, any one with binocular vision can at once tell whether it has fallen nearer to his eyes or further away from them than the thread. If binocular vision be absent, a few trials show that the relative distances of the falling object and thread cannot be appreciated.

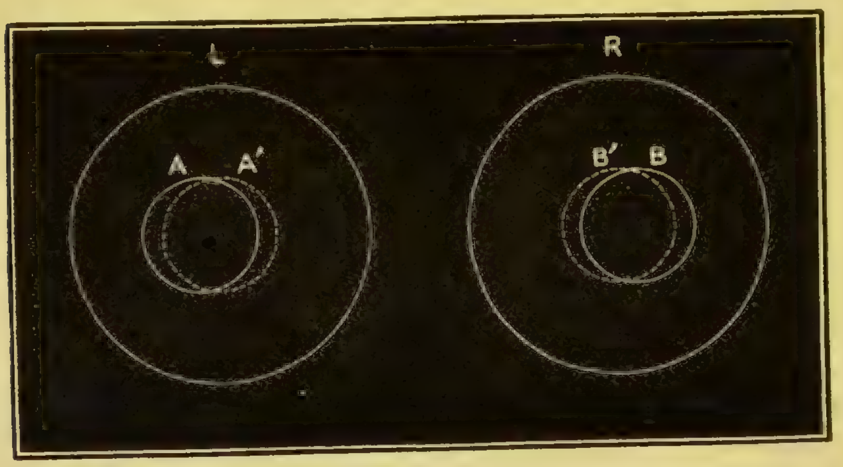


FIG. 6.—Changeable stereoscopic test for binocular vision.

FURTHER OBJECTIVE EXAMINATION.

The examination of the eye by oblique focal illumination is of the utmost importance, and should always be undertaken before making an ophthalmoscopic examination. It is done in the following way. The rays from the source of light used for ophthalmoscopic examinations are concentrated by means of a convex lens on the cornea. By slight movements the focus of light is made to fall on different parts of the cornea, and thus show up any imperfection. By its means any cicatrix, however faint, can be detected. By approaching the lens slightly to the eye the surface of the iris may be brilliantly illuminated, and more thoroughly examined than by mere inspection without the concentrated light. When there is any infiltration or deposit of any other kind in the cornea which calls for closer examination, the oblique examination may be combined with some method of magnification. If the observer be shortsighted, he may be able to approach sufficiently near to obtain a good large image; but if not, another convex lens may be held in the other hand, and the illuminated cornea observed through it. A rectangular portion of a large lens is very suitable for this purpose, as it admits of the structures being seen binocularly. A better idea is thus got of their relative depths. For the more minute examination of corneal affections it is customary to use a strong convex glass placed behind the ophthalmoscope.

The action of the pupil, and the degree to which it contracts to light, may be tested by reflecting light on the eye by means of the ophthalmoscope mirror. In this way the light can be flashed on and off with a very slight movement of the hand holding the mirror. Its intensity can be altered by altering the distance of the mirror from the eye, or the strength of the source of illumination, which can readily be done if a gas jet be used. By keeping a constant degree of illumination on the eyes, and directing the patient to keep fixing the finger, as it is approached gradually nearer and nearer to the face, the presence or absence of contraction of the pupils, which is normally associated with accommodation and the convergence of the visual axes, is tested. Even in cases where the patient examined is blind, the presence of a relation between convergence and pupillary contraction may be tested by causing him to attempt to look at his own finger as he brings it nearer and nearer to his eyes. This will cause him to converge, and if there exists an associated pupillary contraction it will then be seen.

Examination with the mirror alone.—The central aperture of the mirror is held in front of the observer's eye, while the mirror is given such a direction that the light is reflected into the eye to be examined. As a rule, all that is seen by the observer, whose eye is placed behind the mirror, when light is reflected into the patient's eye with the mirror held at some distance from it, is a yellowish-red reflection from the back of the eye. If there is any detail seen in the red area of reflected light, it indicates that the eye examined is out of focus, either long or short-sighted; in other words, that there is ametropia. On the other hand, if the red reflex is either not seen at all when the light has been properly directed into the eye, or is found to be much duller than under normal conditions, or if it is interrupted here and there by more or less dark spots, the indication thus afforded is, that there is a diffuse or circumscribed opacity or opacities in one or more of the transparent media of the eye. If, on oblique examination, such opacity has not been found to exist in the cornea or lens, it may be inferred that it is in the vitreous. If the opacities have an independent movement, or are what is called "floating," they are at once recognised to be in the vitreous. If the opacity causing the interruption in the red reflex is stationary, it is observed to have no movement independent of the eye, although there is an apparent movement as the eye is moved, which is greater or less according to the position of the opacity. If it lies behind the centre of curvature of the cornea (or more correctly, the image of that point which lies very near the point itself), it appears to move in the opposite direction from the eye. If it occupies a position in front of the centre of curvature of the cornea, it appears to move in the same direction as the eye, while the nearer it is to that point the less does it appear to change its position. Floating opacities are most readily made out by causing the patient to move his eyes rapidly in different directions, and then to keep them steady. The objects thus come into view in the illuminated area. Opacities in the lens may be limited, and can often only be seen if the pupil be well dilated with a mydriatic.

There are *two methods of obtaining an image of the fundus of the eye* with the ophthalmoscope, known as the *indirect* and *direct* methods of examination. The former is generally preferable, and in most cases all that is required; but when an examination by this method does not afford an explanation for an existing visual defect, or when it is desired to study more closely any pathological changes, the direct examination should be made in addition, as by it a much greater magnification is obtained. Special attention should be directed—(1.) to the optic disc, and notice taken of its colour, the size and condition of the larger vessels which lie on it, and the state of the immediately surrounding parts; (2.) to the periphery of the fundus, by causing the patient to look up and down, to either side, and, if necessary, slightly moving one's position so as to bring the most peripheral portions into view; and (3.) to the macula lutea. The examination of the region of the macula lutea is often difficult. This is owing to the greater contraction of the pupil, which takes place when the light is concentrated on the macula. In addition there is a disturbing effect produced by light reflected from the cornea. It is generally best not to ask the patient to look directly at the ophthalmoscope, but slightly to one side, and then by slowly moving one's head bring the macula into sight. In this way a view of it is often more easily obtained. Often, however, it is impossible to get a satisfactory view of the macula without dilating the pupil. When the appearance met with at other parts of the fundus is not sufficient to account for the symptoms, there should be no hesitation about using a mydriatic in order to be able to explore the macula properly. It should be a rule never to leave a case without having seen it thoroughly.

The best mydriatic for the purpose is homatropine, as it does not produce such a prolonged paralysis of accommodation as atropine, though it has the disadvantage of acting considerably more slowly. It is also safer in the case of old people, in whom, if there is a tendency to glaucoma, an acute attack of that disease is more likely to be set up by atropine than by the weaker mydriatic. The effect of homatropine may be hastened by combining it with cocaine.

By the *indirect method of examination*, a magnified inverted image is obtained of the structures at the back of the eye. The observer, placing the back of the ophthalmoscope mirror in front of his eye in such a manner that he looks through the aperture in its centre, reflects the light from a lamp or gas jet at the side of the patient's head, into the patient's eye, and from a distance of 12 to 18 inches. With the other hand he holds a convex lens of a strength of from 10 to 15 dioptres, in such a manner that its centre comes to lie in a line joining the patient's pupil with his own pupil, and at a distance from the patient's eye about equal to the focus of the lens. The same lens should always be used in making examinations, so as to give a uniform idea of the relative sizes of the different pictures obtained. To steady the lens in this position, it is customary to rest the little finger against the patient's forehead. There is then formed an inverted aërial image of some part of the back of the eye between the convex lens and the observer, which the observer can see if his eye is properly accommodated for the distance. By moving the head back and forwards, the position in which the image can be most distinctly seen is soon found. It will be further away the greater is the distance of the observer's near point. Hypermetropes and presbyopes generally aid their accommodation by bringing a small convex lens behind the aperture of the ophthalmoscope.

By the *direct examination* a magnified erect image of the back of the eye is obtained. The observer keeping his eye behind the aperture in the ophthalmoscope, reflects the light into the patient's eye from a distance of less than two inches, and receives the rays which pass back from the patient's eye into his own, and, focussing them on his retina, obtains an image of the structure from which they have been reflected. If the observer be emmetropic, and the patient emmetropic and unaccommodated, the image is obtained by the eye without any accommodation. If, on the other hand, the patient's eye be hypermetropic, the observer will have to exert a proportionate amount of accommodation, or place a convex lens sufficient to correct the hypermetropia behind the aperture in the ophthalmoscope in order to obtain a clear image of the fundus. If the patient or the observer be myopic, the latter will only obtain a proper definition of the image of the fundus by making the examination through a concave lens of sufficient strength placed behind the ophthalmoscope. It follows, too, that if the observer be myopic, and the patient hypermetropic, or *vice versâ*, no lens or accommodative effort will be required, if the amount of the defects neutralise each other; while, if they do not, the glass or accommodative change corresponding to the difference in the degree of ametropia is required to obtain a distinct image. This is more fully explained in the chapter on refraction, and cannot be properly understood unless the reader has some knowledge of the errors of refraction commonly met with.

Both with regard to illumination and magnification, the fundus of the eye is seen in a very different manner from any other part of the body. It is this which in great measure causes the difficulty of interpreting what one sees. It is customary to make use of the disc as a convenient measure with which to compare the size of any change seen, or to gauge the distance separating such changes from each other, or from any point (often conveniently the disc itself). Thus we might talk of some hæmorrhage or other spot in the retina being about half the diameter of the disc in size, and situated rather more than two diameters of the disc below it.

The relative depth of the different parts of the back of the eye, seen with the ophthalmoscope, can be estimated with tolerable accuracy by noting the number of the lens which the unaccommodated eye requires to obtain a clear definition, and allowing roughly 3 dioptres to 1 millimetre. By the indirect examination differences in depth are rendered apparent by the parallactic displacement which takes place when the convex lens is given a slight movement from side to side. The structures lying nearer to the eye then appear to glide over the deeper-lying parts as their image moves more rapidly in the same direction as the lens.

A fairly good stereoscopic view of the fundus is got with the binocular ophthalmoscope of Giraud-Teulon, which is so arranged by means of two rhomboidal prisms, the angles of which are 45° and 135°, meeting at their acute angles behind, and at the centre of the aperture of the mirror, that the aërial image can be seen with both eyes at the same time.

Many forms of ophthalmoscope are in use, differing from each other mainly in the kind of mirror used, and the arrangement for bringing different lenses behind the aperture. For estimating refraction, it is of advantage to be able to bring any lens behind the mirror without losing sight of the fundus, and this can be done with most modern instruments. But there is a tendency to multiply the number of lenses and introduce refinements in the mechanism of hanging the mirror, &c., which only add to the cost, without in the least increasing the practical usefulness of the particular ophthalmoscope. The difference in the focal strength of the weaker lenses should not be less than one dioptre.

NORMAL FUNDUS OCULI AS SEEN WITH THE OPHTHALMOSCOPE.

A great variety of different ophthalmoscopic pictures are presented by altogether normal states of the back of the eye. Indeed, the art of ophthalmoscopy consists quite as much in recognising the physiological varieties met with, and in diagnosing the healthy fundus, as in understanding the nature of any pathological changes which may be present. It is usually a comparatively easy thing to recognise the different diseases of the fundus oculi, often very difficult to feel sure that there are no pathological changes.

The *colour* which the fundus presents, and the amount of detail which can be made out, besides, of course, depending to some extent on the illumination and magnification of the ophthalmoscope, vary according to the density of pigmentation in the hexagonal cells of the retina. The red colour comes from the vessels of the choroid, but is modified by the pigment in the retinal hexagonal cells. The degree of pigmentation, again, bears a pretty close relation to the pigmentation elsewhere, so that a different appearance is usually presented by the eye of a very dark individual when compared with that of a light-haired one. In the two extremes, viz., the eye of the negro on the one hand, and of the albino on the other, the difference is very pronounced.

Apart from the actual depth of colour, it is usual to find a want of absolute uniformity of colour. The three regions where one most frequently notices differences in this respect, are the periphery, the area immediately surrounding the disc, and the macula lutea. In the periphery there is often more or less absence of pigment, causing a lighter colouration and greater visibility of the subretinal structures. Round the disc, on the other hand, a lighter colouration is often observable, owing to light reflected from the nerve fibre layer. In the region of the macula again, where the pigmentation is greater, there is always an increased depth of colour. A certain regular unevenness in the colour of the fundus throughout, giving rise to a stippled appearance, is more or less recognisable in different cases by the magnification obtained on direct examination.

Ophthalmoscopic Appearances of the Retina in Health.—The retina, with the exception of its larger blood-vessels, or more correctly, of the blood column in the vessels, is so transparent that in most parts it is practically invisible. In some eyes there is to be seen, and particularly in the neighbourhood of the macula, a kind of dull sheen of reflected light from the surface of the retina. In many eyes, too, there is a more or less marked striation visible, stretching from the disc for some distance in all directions, but mostly upwards and downwards. This striation is due to the presence of the nerve fibres which spread over the retina, and which in this position form a thicker layer than elsewhere.

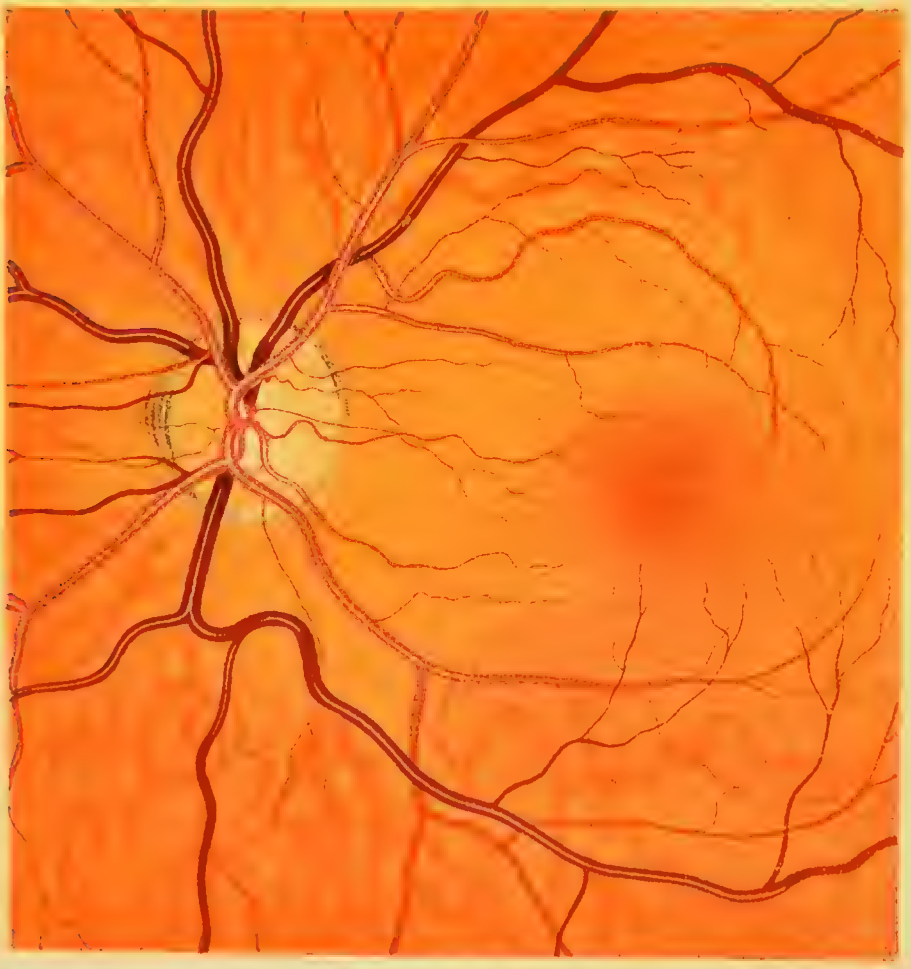


Fig. 7.—Normal Fundus.

The *macula lutea*, or yellow spot, is an area occupying the centre of the retina in the direct line of vision, its centre or fovea centralis being that portion in which the visual acuity is greatest, and on which, therefore, the images of objects directly "fixed" fall. The macula is recognised by its occupying a position to the outer side of the disc, its centre being about two diameters of the disc from the disc margin, by its being relatively devoid of visible blood vessels, and by its darker colouration. Sometimes it is seen to be circumscribed by a bright glittering band or "halo" of reflected light. At the centre of this halo, which is more or less distinctly elongated in a horizontal direction, lies the *fovea centralis*, a

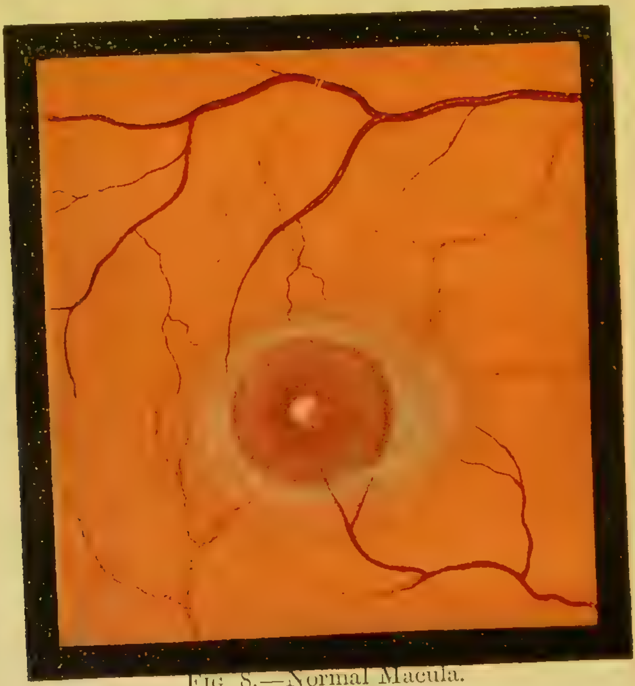


FIG. 8.—Normal Macula.

whitish, pinkish-white, or straw-coloured spot, often presenting at the same time a very marked glitter. The horizontal diameter of the macula is generally distinctly greater than the diameter of the disc; its vertical diameter, on the other hand, perhaps hardly as great as that of the disc. The deeper pigmentation is sometimes pretty sharply defined, but more often gradually fades off into the colour of the surrounding fundus. The yellow colour of the macula is never seen, owing, no doubt, to the preponderance of yellow rays in the light used for examination. The halo round the macula is mostly seen in children, and especially in deeply pigmented retinæ, and hypermetropic eyes. The band of reflection producing it gradually fades off to both sides, sometimes sending indefinite streamers of reflection beyond, particularly to the outer side, or that furthest from the disc. It is, however, usually pretty sharply defined along the edge lying nearest the fovea, where it borders the more deeply pigmented area. Often a complete halo is not seen, but only a sickle-shaped portion of it. The concavity of the sickle then appears on indirect examination turned towards the middle line, though this may depend to some extent on the position given to the convex lens. The reason why the ring is not seen in the same way on direct examination is partly because of the different direction of the illuminating rays, and partly because the illumination is feebler. With a short focus concave mirror there may be more or less indication of it on direct examination in cases where it is marked in the inverted image.

Often at other parts of the retina, too, in young individuals, reflections similar to, though less defined, than that round the macula, are seen as bands of light beside and between the larger vessels. The appearance of these reflections is not unlike watered silk. It is most marked in hypermetropes, and most distinct if the pupil be not dilated.

The appearances described above as retinal reflections are produced by regular reflection from the surface of the retina, the refractive index of which differs from that of the vitreous. The light by which the various details of the ophthalmoscopic picture is obtained, on the other hand, is the irregularly reflected or scattered light from the fundus. Different conditions determine the extent and distinctness of the retinal reflections, viz., the macular halo, foveal glitter and the bands of light along and between the larger vessels. They depend upon the contour of the retina at different places, upon the extent to which the pupil is filled by the rays reflected into the eye (or the shape of the area of illumination of the pupil), upon the intensity of the illumination, and probably also upon individual differences in the proportion between the refractive indices of the vitreous and retina. The intensity of illumination depends not only upon that of the source of light, but also upon the size of the pupil; while the configuration of the area of illumination of the pupil varies with the form of ophthalmoscope used, and the manner in which the light is reflected by it. Most of the regularly reflected rays of light from the retina of the eye examined do not leave the eye in such a direction as to pass through the aperture of the ophthalmoscope, and thus enter the observer's eye. It is only, therefore, from those parts of the retina, the contour of which is such as to give rise to images from which rays pass through the pupil sufficiently near the observer's line of vision, that light reflections are seen. Various explanations have been given of the manner in which these reflections are formed from supposed irregularities in the surface of the retina. It is only recently, and mainly by Dimmer, that the appearances have been carefully studied, and satisfactorily explained in accordance with actually observed anatomical peculiarities in the configuration of the retinal surface. He has shown that the halo surrounding the macula is caused by regular reflection from a circular elevation in this situation produced by a greater thickness of the nerve fibre layer. It is owing to individual differences in the size and completeness of this circular elevation that differences occur in the form of the reflection from it, even when the other conditions which render the reflection visible are equally favourable. The glittering spot at the fovea is the visible portion of an image of the area of illumination of the pupil (or, more correctly, of the reflecting area of the ophthalmoscope mirror), formed by the short focus reflecting concave surface of the central pit or depression. Many of the larger vessels of the retina extend somewhat beyond its inner surface, and this gives rise to concave cylindrical surfaces of short focus. It is by reflection from these surfaces that the bands of light following the vessels, and which are often seen to lie in a plane appreciably nearer the eye of the observer than the vessels themselves, are caused.

The *vessels of the retina*, which appear as darker lines on the reddish fundus, are seen to spring from the disc, or intra-ocular termination of the optic nerve. The arteries and veins are distinguished from each other in three ways, viz.:—(1) by the diameter of the blood column, that is to say, by their apparent relative sizes; (2) by their colour; and (3) by the manner in which they reflect light. The column of blood in the arteries is narrower than that in the corresponding and accompanying veins. The colour of the arterial blood is lighter, and, lastly, a very broad band of light reddish reflection is seen running down the centre of the artery. A streak of light is seen on the veins as well. It differs from that on the arteries in being whiter and much narrower. It cannot be traced so far along the course of the veins, and is often seen to be interrupted at places. The distinctness of the line is greater in both arteries and veins, the stronger the illumination of the fundus. The larger the pupil, therefore, the more distinct does the appearance become. An enlargement of the pupil produces, besides, an increase in the breadth of the line on the veins, but does not lead to any similar alteration of that on the arteries.

The light streak on the veins is in all probability caused by reflection from the convex surface of the blood column, the refractive index of which differs from that of the transparent walls of the vessels. The line on the arteries has usually been referred to the same cause, its greater distinctness being supposed to be due to greater difference in the refractive indices of the arterial blood and the retina. It has, however, been pretty conclusively shown recently by Dimmer that this is not the case, but that it is caused by reflection from the blood-corpuscles of the axial current.

The explanation offered above to account for the light streak on the veins was first given by V. Jaeger, and was by him assumed to hold good in the case of the arterial light streak as well. The following points have been urged by Dimmer in proof of the correctness of V. Jaeger's explanation as far as the veins are concerned:—If the illuminated pupil be taken as the object whose image is reflected from the convex cylindrical mirror formed by the blood-column, then the streak has the breadth which it should have if it be caused by the rays, which, passing from the reflected image through the pupil of the eye examined, are seen by the observer. It increases in breadth, too, as it should then do, on the enlargement of the pupil. The line of light is white, just as it is found to be in experiments where a reflection undoubtedly does take place from the convex surface of a column of liquid. It is too narrow to be produced by reflection from the corpuscles contained in the axial blood-current. In dogs, the broad streak which is seen on the veins under normal conditions of circulation is changed into a much narrower streak when the blood-current is arrested by ligaturing the optic nerve. The narrower streak, which then takes the place of the broad one, corresponds in breadth to what it should be from calculation.

On the other hand, the reasons for believing the light streak on the arteries to be the visible expression of their axial blood current, are, according to Dimmer, that it is three times as broad as it should be were it due to reflections in the same manner as the streak on the veins, but it corresponds in breadth to the streak found in the retinal veins of dogs, which the experiment of ligaturing the optic nerve shows to be caused by the axial current, and, lastly, that the colour of the streak is distinctly red. The red colour is caused by the transmission of the light reflected from the axial portion of the blood column, through the more slowly moving peripheral layers. Why it is that the axial current should be visible in the veins of dogs, and not in man, is probably sufficiently explained by the darker colour of the human venous blood.

The arteries, besides being narrow, are more tortuous than the veins. Not unfrequently they are even seen to twist round the veins at places. This twisting of the arteries round the veins, as well as the sharp turning of the veins, which often takes place as they emerge from the central depression on the disc, gives rise to an alternate, visible, partial emptying and refilling of small portions of the larger trunks on the disc, synchronously with the heart's action. This, which is called *venous pulsation*, is a very common physiological phenomenon, not to be confounded with visible pulsation in the arteries, which is probably always pathological.

The main trunks of the central artery and vein divide on the disc, or more frequently, some time before they come into view, into a superior and an inferior branch. The further division which takes place is always dichotomous, the narrowing becoming gradually more and more appreciable as the vessels pass to the periphery. The situation in which the main division of the vessels takes place on the disc, first into an upper and lower branch, and then each of these into a nasal and temporal portion, is subject to such great variety, that it is almost impossible to find two eyes with exactly the same arrangement in this respect. Even the number of vessels on the disc is not constant. The site at which the first division takes place may also produce some differences in the ophthalmoscopic appearance. Thus, when the artery or vein divides just on a level with the disc, having up to this point coursed in a direction parallel with the axis of the nerve, the appearances presented as one looks down, as it were on the cross section of the main vessel, and therefore on a perpendicular column of blood, is that of a much darker spot at the point of division.

Sometimes, indeed by no means seldom, vessels are found to the outer side of the disc, which are seen to spring into view at its margin, between it and the edge of the choroidal ring. These vessels, in the first part of their visible course, are usually directed towards the disc. After making a more or less extensive curve they pass into the retina. They appear sometimes to be offshoots of the central vessels which have passed out of the nerve before reaching the disc. They are more frequently, however, branches of the short posterior ciliary arteries, and have received the name of *cilio-retinal vessels*.

Ophthalmoscopic Appearances of the Optic Nerve in Health.— The optic disc, or the end or cross section, as it were, of the

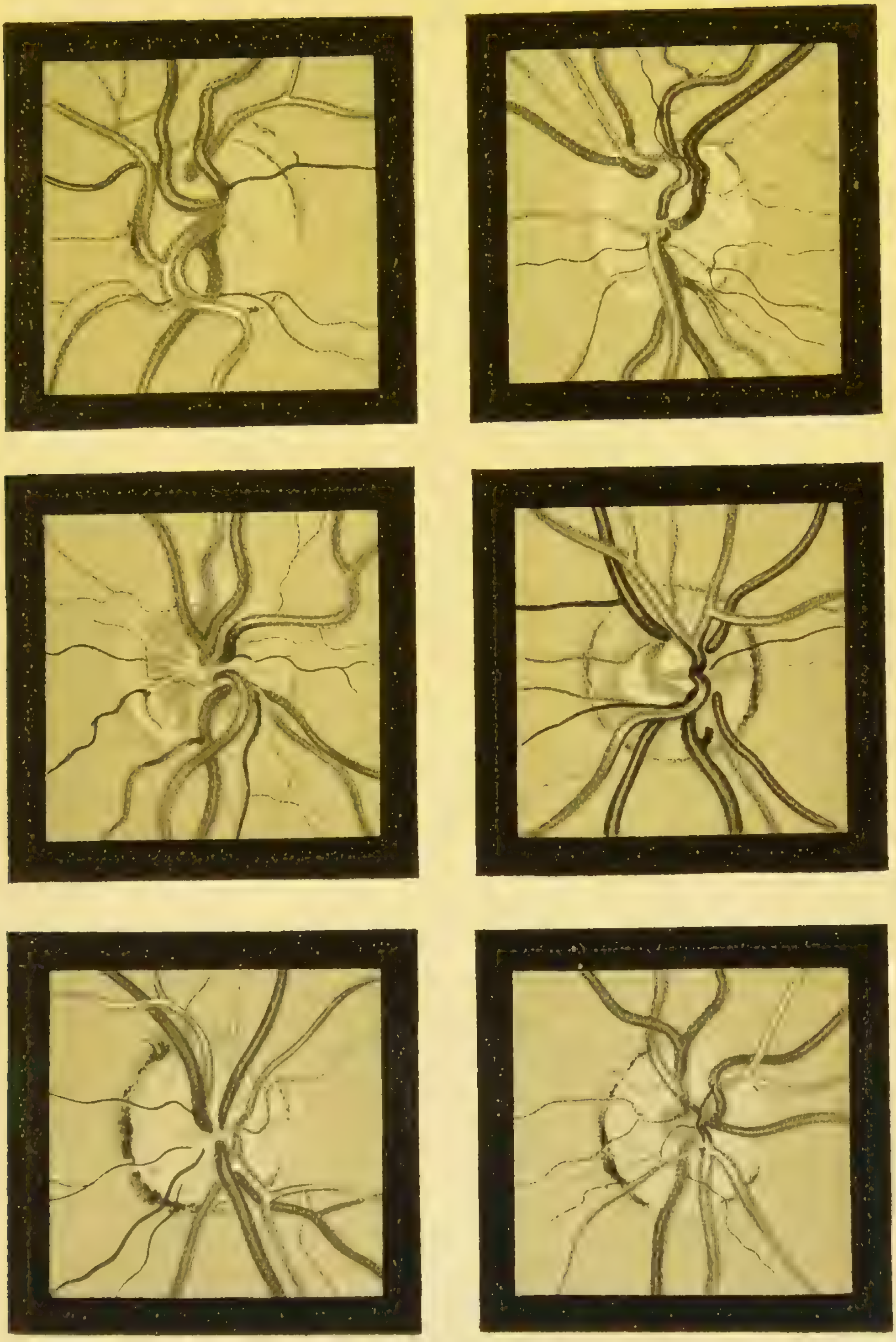


FIG. 9.—Normal discs, showing different arrangement of vessels, &c.

optic nerve, that portion which is alone visible with the ophthalmoscope, receives its name from its circular *shape*. Its real diameter varies in different individuals from 1·5 to 1·75 mm. There is often, too, a difference according to the direction in which the measurement is made. It is, in fact, rarely a very perfect circle, though, in the majority of cases, it is very approximately circular. Considerable differences are met with: thus it may be very decidedly elongated in one direction, most frequently obliquely, but with the long axis nearer the vertical than the horizontal. It is sometimes flattened more or less at one or more parts. It is important that the natural oval shape of the normal disc, in some cases, should not be confounded with the oval form seen, owing to purely optical conditions, where there is astigmatism. In order to convince ourselves that the oval form is that really presented by the intraocular end of the nerve, we have only to withdraw or approach the convex lens, and observe that no alteration of shape takes place.

The disc is far from having a uniform colouration, and the *colour* is one which is not very easily described. It is subject, besides, to considerable variation. This should be remembered, and great care exercised not to infer too readily that an apparently excessive degree, either of pallor or hyperæmia, is pathological. At first sight it is markedly pale or white as compared with the rest of the fundus. What gives the white appearance is the reflection of light from the lamina cribrosa, the altered sclerotic coat through which the nerve passes. But a closer inspection shows that the disc is not by any means colourless. It presents a rosy-pink colouration, which at some parts appears as if mixed with grey, at others with blue, and through which, one, as it were, sees the white light shining. The colour varies according to the degree of illumination, and is less uniform in a feeble or moderate illumination than in a very strong one. The differences in colour, too, are most clearly seen on direct ophthalmoscopic examination. The most markedly pinkish portion is almost always a greater or less crescentic bit of the inner or nasal half of the disc, that portion which is farthest away from the macula. This part is often so decidedly coloured that it presents but a slight difference from the surrounding fundus when the illumination is not great. There is always, however, an appreciable difference in hue.

The pinkish colour of the disc is due to the reflection of light from the blood in the vessels which supply the intraocular end of the nerve; and it is owing to the layer of nerve tissue being thicker, and consequently occupied by a greater number of blood vessels to the inner side than elsewhere, that the colour is deeper in that portion. Less light penetrates through to, and is reflected from, the underlying lamina cribrosa than in other parts, where there is a thinner layer of nerve fibres. The least coloured, and often decidedly, even intensely, white portion of the disc, is its centre. At this spot, owing to the separation of the nerve fibres in all directions, there is a little conical, funnel-shaped depression formed, at the bottom of which there is nothing but the lamina cribrosa, the light reflected from which does not suffer any alteration before it meets the eye. The result is, therefore, that one sees a white circular spot in this situation, which sometimes, on direct examination, exhibits some indication of the structure of the lamina cribrosa. Often the white area is not circular, but while very markedly white, and contrasting strongly with the bordering coloured portion to the inner side of the disc, merges more slowly into a deeper colouration in other directions.

Not unfrequently starting from this point, a sector-shaped area of the outer or temporal portion of the nerve is very devoid of any pinkish colour, and presents instead a mottled bluish-white aspect. This mottled appearance is due to reflection of light from the lamina cribrosa, and the nerve bundles which pass through that fibrous meshwork. The strong dull white is caused by reflection direct from the fibrous tissue, the steel-coloured, bluish-white, more diaphanous appearance, on the other hand, is the result of repeated reflections from the pits or channels in the connective tissue through which the transparent nerve fibres pass. This appearance of the outer half of the disc, which is sometimes seen, appears to depend more on the absence of blood vessels, than on the absence of nerve fibres in that region, although they undoubtedly form there a shallower layer than in other parts.

Occasionally masses of pigment, which rarely attain any size, are seen in the disc. These pigment spots are mostly formed near the margins.

The *margins of the disc* are always sharply defined, though sometimes this definition is less marked above and below than elsewhere. It is usually seen to be surrounded by a white or slightly yellowish-white ring. This is what is called the "scleral ring." It varies considerably in breadth in different cases, but is most frequently very narrow, only just discernible on careful examination by the direct method. The scleral ring is formed, as its name implies, by a surrounding portion of the sclera, the white colour being due to the strong reflection from that coat. The opening in the choroid is always larger than that in the sclera, and hence it is possible to see this portion of the sclera; although, owing to the overhanging of the expanded intraocular end of the nerve, as well as to the frequent heaping up of pigment at the margin of the opening in the choroid, the view of the sclera in this situation is often very much masked. The portion of the ring to the outer side of the disc is commonly the most evident. It is in this direction too, mostly, that it is usual to find some pigment accumulated at the margin of the choroidal opening or "choroidal ring."

Physiological Excavation of the Disc.—One of the most important and frequent variations from the typical condition of the disc, is that to which the name of physiological "cupping," or "excavation," of the disc is given. This variety in the conformation of the intraocular end of the nerve, is met with in very different degrees and in different forms. Altogether, some degree of cupping is about as frequent as the absence of any cupping at all. Although they vary thus greatly, all physiological excavations have the following characteristics in common: they never occupy the whole area of the disc, and they never extend deeper than to the lamina cribrosa. Further, the condition is always met with in both eyes, though not always to the same extent. These points should be remembered in distinguishing a physiological from a pathological excavation —not always such an easy matter as might be supposed.

The physiological excavation is merely a broadening out of the normal depression met with at the centre of the nerve. The sides, or some part of the sides, of this widened depression are sometimes steep, even occasionally overhanging, at other times sloping. The result of a steep cupping is, that on following the vessels along the disc, towards its centre, they are seen to suddenly come to an end, owing to the straight dip which they make down the side of the excavation. They reappear at the bottom of the excavation, where they are not in focus at the same time with those on the surface of the disc and retina. At the same time, the deeper lying portions often look as if they were not continuous with the others, owing to the change in direction which has taken place in the portions hidden from view. The white area of excavation contrasts very markedly with the pink surrounding portion of the disc. The vessels in this white area which, when those on the retinal surface are in focus, look paler, as it were washed out, and on which the clear streak of reflection is absent, assume a more normal appearance on accurate focussing. At the same time, the indefinite white or bluish-white area on which they lie also comes into focus, and shows more or less of the reticulated steel grey, or blue and white meshwork of the structure of the lamina cribrosa. The mottled bluish marking is very different in different cases, as it is only seen when the nerve fibres have lost their myeline sheath at some distance back. In the more funnel-shaped excavations, the vessels may be traced down the side of the cup.

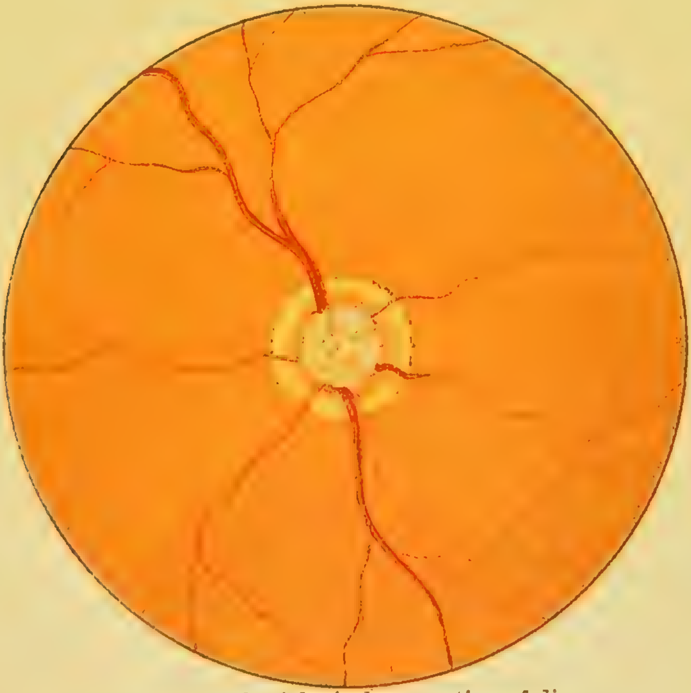


Fig. 10.—Physiological excavation of disc.

In all cuppings, but most markedly in the sharp ones, the vessels at their points of flexure appear darker, owing to one's looking down upon a deeper column of blood. However sharply the vessels may bend over the margin of the cup, they are always seen to course over some portion of the normal disc before passing into the retina.

Ophthalmoscopic Appearances of the Normal Choroid.—When the pigment in the hexagonal cells of the retina is abundant, all that can be seen of the choroid is the red or orange-red colour, which the blood, in its numerous blood-vessels and capillaries, gives to the fundus of the eye. The intensity of this colour, too, is, as has been said, dependent upon the density of the pigment, so that the red is much less pronounced in very dark individuals. Where the pigment in the hexagonal cells is scanty, on the other hand, the vessels of the choroid come into view. As the larger trunks of the choroidal veins lie towards the equator of the eye, the structure of the choroid is often more visible at the periphery than at the centre, where too the pigment is usually more dense. On this account, it is comparatively rarely, under normal conditions, that much of the choroid is seen at the centre of the fundus.

The *choroidal veins*, when visible, present themselves as a dense network of broad and narrow stripes, of a pale red, orange, or yellow colour, separated by lighter or darker interspaces, according to the amount of pigment contained in the stroma of the choroid. If there is a great absence of pigment, both in the hexagonal cells and in the choroidal stroma, the veins of the choroid may be visible in their finest ramifications, or, at all events, as far as the magnification of the ophthalmoscopic image admits of. The interspaces then appear non-pigmented, and reflect a yellow or reddish-yellow light. They vary in size, and their shape is mostly that of an irregular rectangle. The interspaces are more elongated towards the periphery than at the centre, in the region of the macula.

Sometimes the choroid itself is well supplied with pigment, although that of the retina is sparse. The interspaces are then darkly pigmented, and only the larger vessels are visible.

A zone at the extreme periphery of the choroid which lies anterior to the equator of the eye, cannot be seen with the ophthalmoscope.

Senile Changes in the Fundus.—In addition to the great physiological varieties and congenital anomalies which are met with in the fundus, there are often to be seen certain marked senile changes, which are not necessarily of the nature of disease. Thus the optic disc may lose its semi-transparency, and become more or less muddy or dull in appearance, without any pathological change being thereby indicated. Depigmentation of the retina, which may occur in patches here and there, admitting of the structure of the choroid coming into view, is common as a senile change. A degree of atrophy of the choroid, presenting itself in a broad band of a dirty yellowish or straw colour surrounding the disc, is often seen in old people.

CHAPTER II.

DISEASES OF THE EYELIDS AND LACHRYMAL APPARATUS.

Diseases of the Eyelids.

THE obvious function of the eyelids is to protect the eyes from external injury, and thus preserve the transparency of the cornea. The lids, with their lashes or cilia, prevent substances of any size coming in contact with the cornea, and furnish an oily secretion which lubricates the surface, and thus obviates any intransparency of the cornea which might arise from the drying or hardening of the superficial layers of its epithelium. The tears, too, aid in this, and at the same time tend to wash away any minute substances which may lodge within the eye.

A few points in connection with the anatomy of the lids may be here referred to, as it is necessary to be acquainted with these in order to understand the pathology of the diseases which affect the lids, and to follow the descriptions of the various operations which are performed on them. The lid aperture varies considerably in length in different individuals, and always appears larger when the eyes are prominent, and smaller when they are sunken. The points where the upper and lower lids meet at an angle are called the outer and inner *canthi*. Rather less than a quarter of an inch from the inner canthus, and just at a point where the lid margins begin to narrow, are situated, both above and below, the slight papilliform elevations in which the little channels or *canaliculi* leading to the tear sac end. The small punctiform apertures leading into the canaliculi are the so-called *puncta lachrymalia*. The eyelashes spring from the outer portion or edge of the margin of the lid; their roots, which are from 2 to 3 mm. long, being embedded in the dense fibrous tissue.

The substance of that portion of the lid which covers, or is directly applied to the eye, is composed of four layers, which, proceeding from within outwards, are (1) the conjunctiva; (2) the tarsus; (3) a layer of muscular fibres; and (4) the skin. In this situation, the conjunctiva is very closely adherent to the underlying dense fibrous tissue or *tarsus.* Beyond this, it is thrown into folds, and only supported behind by a much looser connective tissue. The tarsus of the lower lid is an insignificant, narrow, and thin structure; in the upper lid it is thicker and much deeper, and to its free margin is attached the tendon of the levator palpebræ superioris. An oily secretion is formed in the tarsal or *Meibomian glands,* and passes out of numerous ducts which open along the margin of the lid. Immediately below the skin of the lid, which is extremely lax and moveable in all directions, is the *orbicular muscle,* the function of which is to close the eyes. This muscle, which is supplied by the seventh nerve, is a large flat structure, composed entirely of voluntary muscular fibres, and extending over the margins of the orbit, above and below. It has tendinous attachments to the inner and outer palpebral ligaments.

The upper lid is raised by the levator palpebræ superioris, which is innervated by the third nerve. To some extent also that muscle receives assistance from a bundle of non-striped fibres, which lie under the conjunctiva, and are attached to the free edge of the tarsus. These fibres are generally called *Müller's muscle;* they also exist in the lower lid, and are innervated by sympathetic nerve fibres.

Blepharitis.—This is one of the most common affections. It occurs mostly in children who are either strumous or ill-nourished, and dirty and ill-cared for. Often it begins after measles, and is not infrequently associated with catarrhal or phlyctenular conjunctivitis, and with inflammation of the lachrymal sac. The inflammation, which is often termed *tinea tarsi,* or *ophthalmia tarsi,* is an eczema of the margin of the lid. It begins with hyperæmia and increase of the secretions. When this is not attended to, the secretion becomes fibrinous and glutinous, and seals the eyelashes together, forming crusts or scales, under which a process of ulceration, leading to destruction of their follicles, takes place. The affection is met with in very different degrees of severity, from what is merely a tendency to slight scaliness of the lid margin, to the formation of marked pustules, the irritation of which causes considerable œdema and inflammatory swelling of the lid. It is very chronic, many cases seen in hospital practice having existed for months and years before any advice is sought. When not treated in time, it leads when severe to complete destruction of the eyelashes. Often a few eyelashes are found, after healing has taken place, to be misdirected, owing to cicatricial contraction round their follicles.

The *treatment* of blepharitis consists in first removing the scales or scabs, and then applying to the raw surface the yellow ointment of Pagenstecher (8 grs. to ℥i. of yellow oxide of mercury, ℥i. of lanoline or spermaceti ointment, mixed with a few drops of olive oil). This ointment should be applied at least twice a day, morning and evening, and is greatly superior to any other preparation of mercury, either the red oxide or the mitigated nitrate. The scabs may be removed with a piece of quill. This can be done without much pain if the surface has been previously soaked for some time with an alkaline lotion (about 5 grs. of bicarbonate of soda to ℥i. warm water). A thorough removal of the scales should be made before each application of the ointment. As a rule, it is only the first time that any difficulty is experienced in doing this, so that when the crusts are thick and copious, it is better for the surgeon to remove them himself. He may then apply a little caustic solution

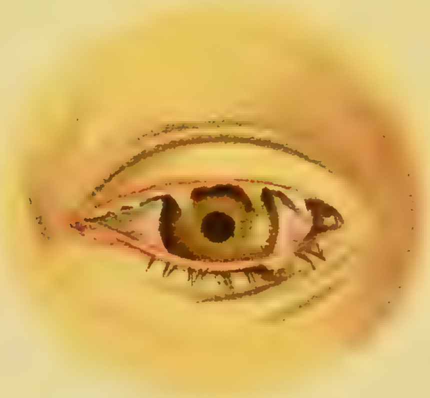


FIG. 11.—Blepharitis.

(20 grs. of nitrate of silver to ℥i.) along the raw surface. Sometimes it is advisable to cut the eyelashes quite short before removing the crusts, or, when only a few remain, they may be epilated. In obstinate cases the condition of the lachrymal sac should be looked to, as it is often necessary to treat it in accordance with the rules laid down at page 75, before the blepharitis can be permanently cured. This will be found almost invariably to be the case where the blepharitis is unilateral, and has resisted local treatment. The state of refraction should also be ascertained. Hypermetropia and astigmatism have certainly some influence in keeping up, though it is unlikely that they actually, as many suppose, give rise to this condition.

HORDEOLUM or STYE is an acute inflammation of the cellular tissue of the lid, leading to suppuration which points at the margin of the lid. Often, like boils elsewhere, it is the tissue surrounding the root of an eyelash which becomes inflamed. The inflammation causes very considerable, often throbbing, pain, and generally a good deal of œdematous swelling of the lid. The swelling may be so great as to almost completely mask the stye itself. Sometimes two or more occur at the same time, and not infrequently there is a tendency for crops of them to appear in succession. The point to be remembered in the diagnosis is the tendency to point at the margin of the lid. In this respect a stye differs from a suppurating tarsal cyst (see page 54), with which it might be confounded. Styes may sometimes undergo absorption without bursting and discharging externally, as is the case with localised inflammations in other parts of the face—for instance, the nose and the ear, where the tissues in which the inflammation takes place are very dense.

The *treatment* consists in applying compresses of lead or alum lotions. If a stye is on the point of bursting, it may be poulticed; but this is not otherwise advisable. The same may be said with reference to incision. When crops of styes appear, a course of some aperient mineral water followed by iron, and the local application twice daily of mitigated nitrate of mercury ointment (one part of the ointment of the pharmacopœia mixed with seven parts of lard) often does good. A generous diet, open air exercise, and change of air, are also indicated.

MOLLUSCUM CONTAGIOSUM is another affection frequently met with along the margin of the lids. It occurs on the skin of the lids as well, and there the cysts may attain a greater size. The circular shape of the swellings, and the little depression in the centre, give to them quite a characteristic appearance. They are often found in several members of the same family. The affection is common in children, and occasionally goes on to suppuration.

The *treatment* consists in snipping off the smaller growths with a pair of scissors, and in transfixing the larger ones, and pressing out the sebaceous matter which they contain; the cyst wall may be drawn out, too, with a pair of fine forceps.

WARTS, PAPILLOMATA, and TRANSLUCENT CYSTS also occur along the lid margin. They should be removed with as little mutilation of the normal tissues as possible, so as to avoid any misdirection of the eyelashes, which is liable to occur from cicatricial contraction.

HERPES FRONTALIS.—This is a rather uncommon disease. The eruption, which is limited to the one side, generally occurs on the forehead and lids, and sometimes the side of the nose as well. When the branch from the fifth, which supplies the side of the nose, is affected, the cornea may become ulcerated, and the iris inflamed. The eruption is preceded by intense neuralgia, which rarely lasts more than a day or two, though occasionally much longer. The blebs, which then appear, are at first clear. After two or three days their contents become muddy; ulceration then takes place, and they eventually leave scars. The scabs fall off in about two weeks from the appearance of the eruption, but the neuralgia may continue, although the skin itself is more or less anæsthetic. I have seen the eruption confined entirely to the upper lid, and in one case it occurred first on one side, and then on the other, after several years' interval. I have also seen recurrence on the same side. Such cases have been described by Nieden as associated with chronic inflammatory conditions of the cervical vertebræ. Herpes frontalis is considerably more common in men, especially old men, than in women, and is said to occur twice as often on the left side as on the right. The disease has been shown to be produced by a lesion of the Gasserian ganglion, but it is doubtful whether this is always the cause.

No local *treatment* is called for. When the neuralgia is severe, subcutaneous injections of morphia are useful. After recovery, the patient should, if possible, be sent for change of air, and the diet should be nourishing.

Xanthelasma.—This is the name given to a yellow patch of irregular outline which occurs in the skin of the lids. It is almost always met with in both upper lids at the same time, though the patch may be much larger and more prominent in the one than in the other. It consists of fibrous tissue containing some altered blood pigment.

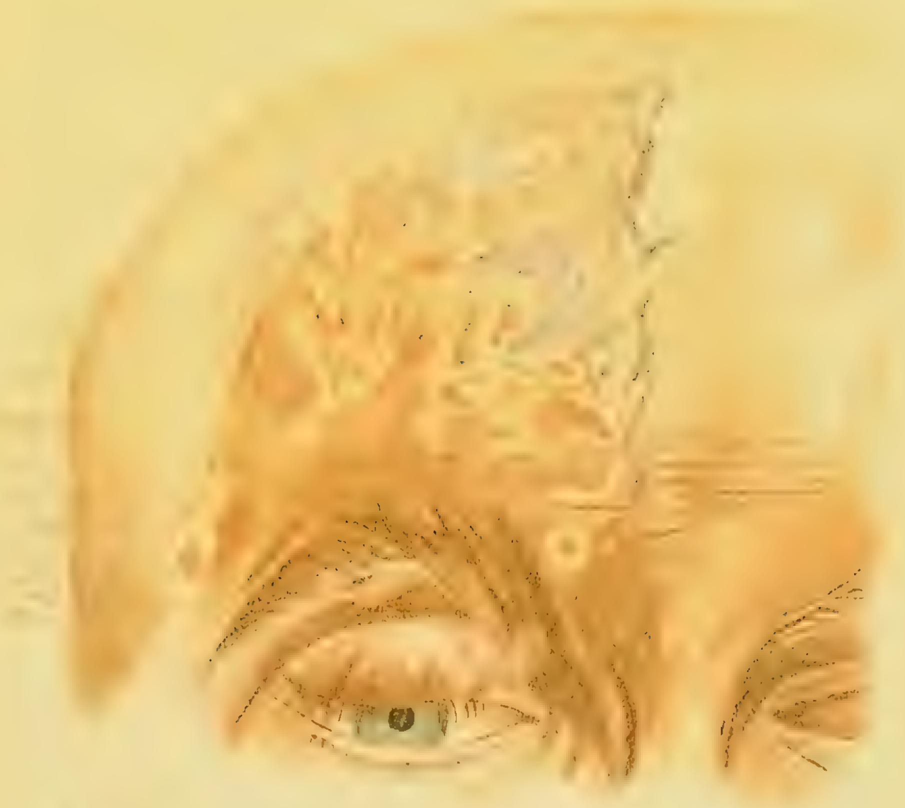


Fig. 12.—Herpes Frontalis.

The patches may be removed without difficulty if the patient wishes it, as is sometimes the case, on account of their somewhat unsightly appearance.

Eczema may extend to the lids from other parts of the face, or begin originally in the lids as an extension from a conjunctivitis or a blepharitis, which, as we have seen, is in reality nothing else than an eczema of the lid margin. Some people are particularly subject to this; thus I have several times seen an acute eczema of the lids set up by compresses of corrosive sublimate solution even in the dilute form (1-5000), in which it is found useful as an antiseptic preparation in various affections of the cornea, and as a dressing after operations on the eye. It is occasionally also set up by atropine, when used as an anodyne and mydriatic, in different affections of the eye. Conjunctivitis in children is frequently associated with eczema, generally due to the practice of poulticing the eyes, which is a common popular remedy, often leading to much increase in the severity of what would be otherwise very slight ailments.

In adults, zinc ointment or boracic acid ointment are as good local applications as any; in children, perhaps nothing is better than Pagenstecher's ointment, the part being well washed first with black soap and water.

MILIUM, or small, circular, white tumours, rarely more than a pin's head in size, are often found in considerable numbers in the skin of the lid and cheek. The epidermis covering them is very thin, so that they are of a pearly white colour.

They are removed by incision, and by squeezing out the compressed and altered epidermis which they contain.

CHALAZION, or TARSAL CYST, is a small tumour situated in the tarsus. It may grow to the size of a small hazel nut, but is rarely much bigger than a good-sized pea. Sometimes a number of these tumours occur at the same time, or at intervals, in the same individuals. The skin of the lid is always freely moveable over them, while the conjunctival surface is more or less involved. The conjunctiva over the chalazion may be merely thinned and reddened, and present a kind of gelatinous appearance from the shining through it of the underlying tumour, or it may be converted into what looks exactly like granulation tissue. Chalazion is a tumour caused by the retention of secretion in the glands of the tarsus, followed by an inflammation of the surrounding tissues. If the inflammation be acute it goes on to suppuration, and such cases may give rise to an appearance not unlike that met with from a stye. The chalazion always, however, points somewhere on the conjunctival surface of the lid, whereas the stye points at the lid margin. When properly removed a chalazion does not recur.

Treatment.—The contents of the cyst should be shelled out through an incision made in the conjunctiva covering it. It is best to make the incision vertical, and therefore parallel with the direction of the glands in the tarsus. It is not advisable, as is sometimes done, to cauterise the cavity left, but a little rough manipulation with a small scoop, after the contents have all escaped, with the object of breaking down the wall, renders the destruction more complete. In a few cases the evacuation can be better made through a small horizontal incision in the skin above, but this is seldom necessary.

Chalky Infarcts in the Meibomian glands are met with mostly in elderly people who have suffered from chronic hyperæmia of the conjunctiva, and are of a more or less distinctly gouty habit. When these little yellow concretions project beyond the surface of the conjunctiva they are apt to set up irritation by scraping against the cornea, and should then be removed.

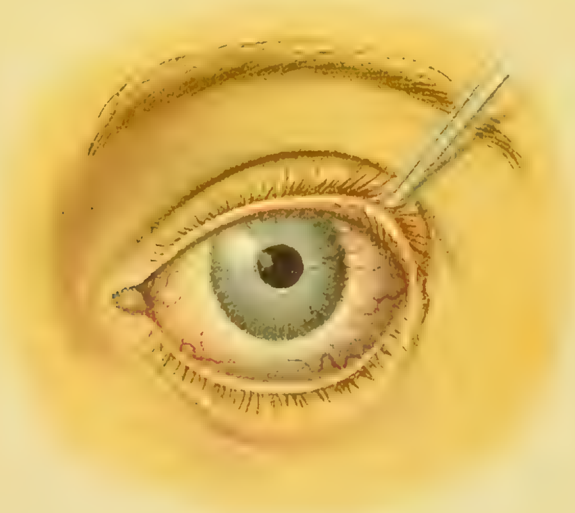


Fig. 13.—Distichiasis.

Trichiasis and Distichiasis.—Sometimes occurring as a congenital abnormality, but more frequently as the result of blepharitis, is a misdirection of the eyelashes, which gives rise to more or less irritation by their rubbing on the cornea. When there is a double row of lashes, the inner of which are misdirected so as to point backwards, while the outer are normally directed, the condition is known as *distichiasis*. Other cases, whether all or only a few of the lashes are misdirected, are cases of *trichiasis.*

Treatment.—When the trichiasis is only partial, a temporary improvement is obtained by epilation. In some cases, where a few eyelashes only have been left altogether, the patient may procure for himself a pair of forceps, and have the eyelashes removed whenever they cause irritation. A radical cure can, however, only be obtained by some operation. The simplest, where a few cilia only are misdirected, is either that called repositio ciliorum, or destruction by the actual cautery, or by electrolysis, of the follicles from which they grow. In more general trichiasis and in distichiasis some operation for the transplantation of the follicles (see chapter on operations) has to be adopted.

ENTROPION.—When the lashes rub against the cornea, owing to an incurving of the lid margin, the condition receives the name of entropion. Of this there are two principal varieties, *spasmodic* or *muscular* entropion and *cicatricial* entropion. The most common source of the first variety, which is mostly met with in the lower lid, is some irritation of the eye which gives rise to spasmodic contraction of the palpebral portion of the orbicularis. Under such conditions the inversion of the lid is favoured by a lax condition of the skin, and a narrowness of the palpebral aperture. It is very often met with in old people after operations, and then constitutes a troublesome though rarely serious complication during the healing process. In some cases, more particularly in children, the irritation of foreign bodies in the conjunctival sac or on the cornea gives rise to the same variety of entropion.

Treatment.—When the condition is evidently only temporary, the lid may be retained for some time in a proper position by means of pieces of plaster, or by painting it over pretty thickly, after drying the skin well, with flexile collodion. In many cases it is necessary to remove a piece of skin from the lid.

In some children entropion is a congenital condition, and then has quite a characteristic appearance. The lashes, though rubbing against the cornea, do so, not with their points, as in other cases, but are so applied as to be parallel with the cornea, and consequently produce little or no irritation, so that the attention of the parents is generally only attracted to the condition on account of the watering of the eye to which it gives rise. This variety of entropion is probably due to an abnormal development of the orbicularis in the vicinity of the lid margin, and is cured by removal of an elliptical piece of skin.

Another common variety of muscular entropion is that met with in cases where the lid has lost the support of the globe, and this may occur in both upper and lower lids, and be a source of irritation to the conjunctiva. It is seen both when the eye is absent altogether and no artificial eye has been worn, and where there is shrinking of the eye, or *phthisis bulbi*, from any cause.

Cicatricial entropion follows loss of substance in the conjunctiva, and may thus result from burns or other destructive accidents, from trachoma and diphtheritic conjunctivitis. The condition is slowly brought about by the cicatrisation in the conjunctiva, which follows such processes. The inverted lashes lead to abrasion of the corneal epithelium, and a chronic vascularisation and consequent intransparency of that membrane, and the entropion therefore calls for some radical cure.

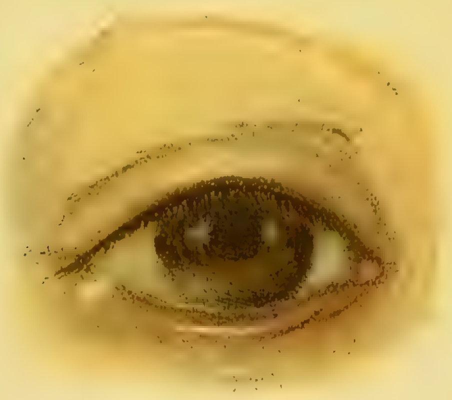


FIG. 14.—Cicatricial entropion (from case of essential shrinking of conjunctiva).

The operations best suited for such cases are those of Hotz, Snellen or Streatfield, Dianoux, and v. Milligen or Benson, which are described in the chapter on operations.

ECTROPION.—That condition in which the lid is everted is called ectropion. The eversion may be only slight—that is, the edge of the lid may merely be drawn away from its normal position against the eye—or it may be so great as to expose almost the whole of the palpebral conjunctiva. Two varieties of ectropion may be distinguished, *conjunctival* ectropion and *cicatricial* ectropion. The conjunctival variety begins as an inflammation of the conjunctiva, the swelling of which, as well as the extension of the inflammation along the tear canaliculi, lead to a separation of the lid margin from the eye, and a consequent interference with the normal excretion of the tears. The ectropion only occurs in cases where the conjunctival inflammation has led to a paralysis of the palpebral portion of the orbicularis muscle from infiltration and distention. When the position of the lid has thus been altered, the tears flow over the cheek, causing eczema, with thickening and excoriation of the skin, so that a vicious circle is set up, which tends always further and further to increase the deformity. This variety of ectropion is shown in Fig. 15.

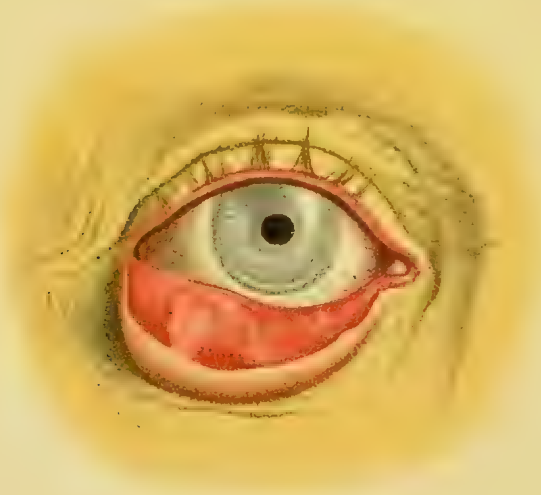


Fig. 15.—Conjunctival ectropion.

Treatment.—In the lower degrees of conjunctival ectropion, all that is required in order to bring the lid back into its normal position, is to slit up the canaliculus with a Weber's knife, so as to establish a better passage for the tears, and at the same time to keep the skin soft with some simple ointment, and touch the conjunctival surface with nitrate of silver in the way described at page 82. When the ectropion is considerable, this process, which leads in any case only slowly to recovery, is not generally sufficient, but must be supplemented by the introduction of sutures after the manner first introduced by Snellen.

In the higher degrees, where the case has been of very long standing, it is sometimes necessary to perform an operation for the shortening of the lid aperture. It is not advisable, as sometimes recommended, to excise any portion of the conjunctiva alone.

A form of conjunctival ectropion is occasionally met with in children, due to the contraction of the orbicularis at a time when the swollen and inflamed conjunctiva has, by some accident, or as the result of some manipulation, become everted. The contraction of the muscle keeps up the eversion, and at the same time leads by constriction to further swelling, so that the ectropion may be difficult to reduce. This condition is most marked and unsightly in the upper lid, and should be carefully guarded against by the application, where there is a tendency to it, of a tight bandage. A slight degree of ectropion occurs, too, in cases of facial paralysis, and causes epiphora. When the epiphora is marked, some operation for the narrowing of the lid aperture is called for.

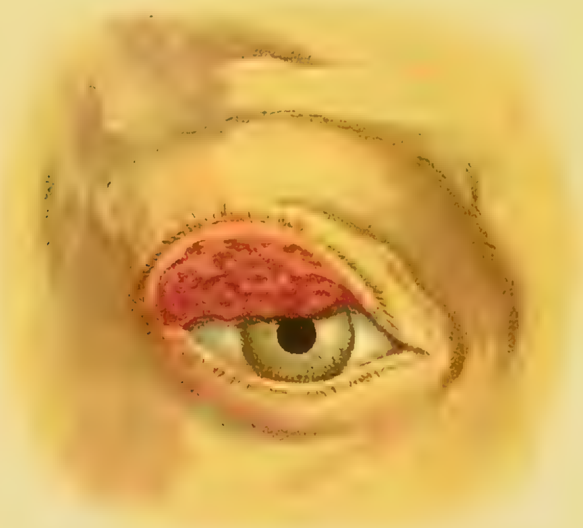


FIG. 16.—Cicatricial ectropion of the upper lid.

CICATRICIAL ECTROPION is produced by contraction following loss of skin of the lids themselves, or of the skin of the face in the neighbourhood. It is met with after lupus, burns, abscesses, &c. Another variety is that which is caused by the tacking down of the skin over the bone of the margin of the orbit or cheek where there has been caries or necrosis in that situation. The absence of skin in cases of cicatricial ectropion usually renders it impossible to remedy the defect by such simple measures as are applicable in conjunctival ectropion. Some plastic operation, or better still, the transplantation of skin from other parts of the body, is therefore necessary. The operations performed for the different forms of ectropion are described in the chapter on Operations.

EPICANTHUS.—In not a few children, a more or less pronounced fold of skin is found covering the inner angle of the lids, and continuous with the fold normally present on the upper lid. This condition is known as epicanthus. When marked, it gives a peculiar appearance to the face, covering, as it does, most of the white of the eyes to their inner sides. As the child grows, and the bridge of the nose develops, the epicanthus either disappears altogether or becomes much less unsightly: so that it is only in high degrees of the deformity that any interference is called for. In such, too, there is at the same time more or less drooping of the lids, which may be associated with a defect in the power of moving the eyes upwards.

An improvement may then be effected by removing an elliptical piece of skin from the bridge of the nose in a vertical direction, care being taken to get union by first intention, by preventing any dragging on the stitches. This method of operating is the old one of v. Ammon. Arlt recommended, and frequently practised, excision of the fold itself on either side of the nose.

PTOSIS.—A not by any means infrequent congenital defect, due probably, in most cases, to a faulty development of the levator palpebræ superioris, is the drooping of the upper lid, or ptosis, which may be present on one or both sides. Sometimes this is associated with a defect in the upward movement of the eye as well. *Congenital ptosis* is one of the hereditary deformities. I have met with a family where it existed in the father and five out of six children. Most cases of congenital ptosis appear to be due to defective development of the levator. Probably some cases, however, are the result of an abnormality in the nerve centre.

Cases have been described, and attention seems first to have been directed to them by Gunn, in which an involuntary raising of the drooping lid has been associated with voluntary movement of the lower jaw. This has usually been on the left side, and has been explained by assuming that the levator is to some extent innervated by fibres springing from the motor root of the fifth. It is certainly not improbable that in the absence of a proper innervation from the third nucleus, this root which lies in such close proximity to it might partially make up for the developmental defect.

The effect of ptosis on vision depends of course greatly on its degree; when the pupils are partially covered, the patient is in the habit of throwing his head back in order to see. When this is the case, and still more when the pupil is covered completely in one or both eyes, some operation has to be performed with the object of raising the lid without interfering with its closing.

A curious form of ptosis, apparently allied to the congenital variety, has been lately described by Fuchs, and called by him *isolated bilateral ptosis.* The name was no doubt suggested by the fact that, with the exception of the ptosis, no other demonstrable affection of the eyes or any other part of the body is found in these cases. The ptosis develops so slowly that the patient cannot say when it has begun. It eventually becomes so complete that no action of the levator is possible. In some cases a slight degree of ptosis is apparently present at birth, and afterwards increases in the same gradual manner. Fuchs has given good reason for regarding these cases as cases of atrophy of the levators. It is doubtful whether the atrophy is myopathic or neuropathic.

Other forms of ptosis met with are such as are due to partial, or, when other muscles of the eye are also affected, complete paralysis of the third nerve—also cases which are merely the result of chronic inflammation of the conjunctiva—mostly trachoma. *Hysterical ptosis,* which may be on one or both sides, is not very uncommon. A peculiar cause of slight drooping of the upper lid, to which attention has been directed by Horner, appears to be from *paresis of Müller's muscle.* It is associated with myosis, and sometimes with diminished intraocular tension, and depends no doubt on some lesion of the sympathetic nerve. Different more or less satisfactory operations for ptosis are described in the chapter on operations.

NICTITATIO.—Constant blinking is often met with, and is known as nictitatio. The cause of this, which is most frequent in children and young adults, is usually some irritation from the eye itself—conjunctivitis or keratitis. Often the irritation originates in some other fibres of the fifth nerve, not uncommonly, for instance, from decayed teeth. It may also proceed from other parts of the digestive tract. I have seen several cases in children, due to worms. At other times the source of irritation is quite obscure, and indeed the blinking is not seldom apparently merely a habit, which has no doubt been originally set up in some such manner as indicated.

An idea of the source of irritation may sometimes be got by dropping a solution of cocaine into the conjunctival sac. If after two or three applications the blinking becomes less frequent, or ceases, it is very probable that the irritation proceeds from the eye; if not, we have to look elsewhere for it—in children, the teeth, or some other part of the digestive system. Often the contraction in the orbicularis is not so general as to cause actual blinking, but is limited to certain of its fibres, mainly those of the lower lid, causing a disagreeable twitch or "twitter" in this region.

BLEPHAROSPASM, or spasmodic closure of the eyes, may be constant or intermittent. The constant cases are associated with photophobia, or the dread of light, and are discussed in the chapter on diseases on the conjunctiva. The intermittent cases are very rare, and mainly caused by irritation of the facial nerves, which causes, at the same time, other contractions of the facial muscles. I have also seen this apparently due to tumours of the parotid gland. These spasms have sometimes been cured by stretching the facial nerve. In some cases of so-called epileptiform neuralgia, there is painful intermittent blepharospasm. In one such case I obtained considerable improvement by tearing the supra-orbital and nasal nerves after all other measures, continued through many years, had been of no avail. In another similar case I stretched and tore both supra- and infra-orbital branches without giving relief.

ŒDEMA OF THE LIDS occurs along with inflammation localised in different parts about the face, and is also frequent in affections of the heart and kidneys, where there is any cause for general œdema. In some people there seems to be a tendency to œdema in this situation, independently of any general cause. This is sometimes called *frigid œdema*, and gives rise to a very heavy appearance of the lids, which can be much improved by removing an elliptical piece of the skin of the lid. Occasionally a persistent œdema of the lids is left after attacks of erysipelas of the face. This may, without danger, be treated in the same way, if the patient desires it, for the sake of the appearance.

Long persisting œdema appears to be associated with chronic conditions of inflammation in the mucous membrane of the nose, to which attention should always be directed when the cause is doubtful.

ABSCESS OF THE LID is most frequently traumatic, often the result of the breaking down of a blood-clot, which has been formed at the time of the contusion. The abscess should be opened and treated antiseptically as soon as fluctuation can be felt. Boils, too, are occasionally met with, principally in the upper lid.

A serious form of infectious abscess of the lid occurs, fortunately only rarely, in children; it begins as a pustule, and leads rapidly to great erysipelatous swelling and redness, followed by gangrenous destruction of more or less of the skin of the lid. The cases I have seen have been exactly like noma elsewhere, and probably it is the same disease. A similar condition is said sometimes to occur amongst butchers and wool-sorters, and often proves fatal.

DERMOID CYSTS are not uncommon in the lids, and are most frequently situated to the temporal side of the upper lid, over the orbital ridge. They can be removed either by being carefully dissected out, or, through a small opening, by puncture, followed by squeezing out of the contents and removal of the sac with a pair of toothed forceps. The incision must be made in either case parallel with the margin of the orbit.

NÆVI OF THE LIDS should be treated by electrolysis, if of any size, or if growing; other methods of treatment are liable to cause too much destruction of the tissues.

RODENT ULCERS and EPITHELIOMATA are not infrequently found affecting the lids, and have to be removed. The loss of substance thus sustained may be made good by some suitable plastic or transplantation operation, but in the case of the lower lid this is often unnecessary, as the deformity, after healing has taken place, is often much less than might have been expected.

Chancres and Vaccine Pocks are occasionally found on the lids, being produced in both cases by direct infection of specific matter. The first, from the considerable time which it takes to develop, is sometimes difficult to diagnose; the vaccine pock, on the other hand, is usually characteristic, and the source of infection readily discovered. Chancre in this situation is probably in most cases communicated by kissing. I have seen it in both adults and children. One of the cases which have come under my observation was a woman of eighty-four, who had charge of an extremely syphilitic infant. Vaccinia is more common in women than in men. Of five well-marked cases which I have seen, four occurred in women. There is always either a distinct history of inoculation, or of the possibility of inoculation. A child has been vaccinated ten or fourteen days before the patient is seen. Usually the pock occurs on the lower lid, but there are also to be found one or more ulcerated patches on the margin of the upper lid where it comes into contact with the macerated surface of the primary vaccine ulcer. The swelling is always great, and involves not only the lids but also the cheek. The base of the ulcer is decidedly harder than the surrounding swelling; but not so distinctly indurated as in the case of chancre of the lid, and the glands are not swollen. There is, comparatively speaking, very little pain; practically no spontaneous pain, and but little tenderness to touch. The eye itself does not seem ever to be affected. Vaccinia of the lid never leads to any alteration in its position, and even the cicatrix left is slight—barely perceptible, owing, no doubt, to the laxity of the skin in this situation.

The syphilitic sore is always a more distinctly clean-cut, eaten-out ulcer, which has taken a considerable time to develop from its first appearance as a pimple at the lid margin. The opposite lid margin is not as a rule ulcerated. The base of the ulcer is greatly indurated; and the pre-auricular gland—often, also, the submaxillary glands—are swollen. There is no history which can in any way connect the case with vaccination, and usually one which renders a syphilitic contagion possible. Secondary symptoms appear in due course.

Sarcoma of the Lid is met with as a great rarity in children and young adults. Other uncommon affections in this situation are *fatty tumours, elephantiasis, neuroma,* and *coloboma.* The last may occur along with coloboma of the iris. It should be treated as soon as possible, by paring the edges of the slit and uniting them by sutures, so as to secure a proper covering for the cornea.

Blepharophimosis — Anchyloblepharon. — Chronic affections of the lids, especially such as keep up an excoriation of the skin at the outer canthus, may lead to a narrowing of the lid aperture, or blepharophimosis. The *treatment* for this is to enlarge the palpebral fissures in the manner described under the name of *canthoplasty,* in the chapter on operations. When either the complete ciliary margin, or a portion of its centre is united with that of the other lid, the condition is known as *anchyloblepharon.* This is sometimes congenital, but more commonly the result of burns or other accidents, causing a loss of substance; sometimes also of long-continued inflammations. The *treatment* is usually simple enough, if it is not associated, as is often the case, with symblepharon.

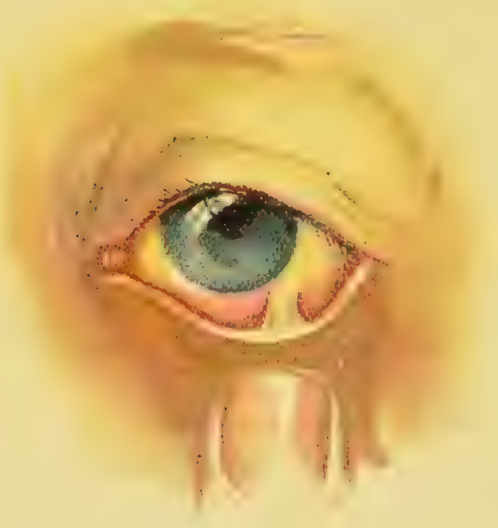


Fig. 17.—Symblepharon.

SYMBLEPHARON.—When some injury or destruction of the two opposing conjunctival surfaces of the lid and eye has taken place, there is a tendency for the lids to grow together, and thus give rise to the condition known as symblepharon. This is most likely to occur in all cases where that portion of the conjunctiva, which forms the fold between the ocular and palpebral coverings, is involved in the ulcerative or destructive processes; indeed, it is then almost impossible, except by performing some operation to effect a covering of the raw surface, to avoid their growing together, as, however frequently the two surfaces be separated, healing will slowly take place from the angle between them.

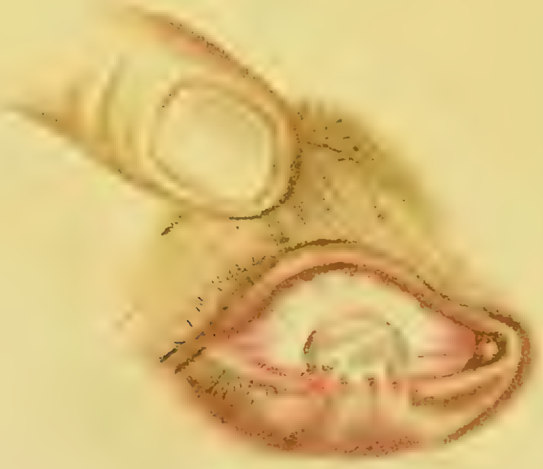


FIG 18.—Symblepharon.

When the fold is not involved, the frequent separation of the opposing surfaces, by making passive movements of the lid, and at the same time dropping a few drops of oil into the conjunctival sac, will generally permit of their cicatrising independently, and thus obviate the symblepharon.

The severity of the alteration caused by symblepharon is very different, according to the position and extent of the adhesion between the conjunctival surfaces. In very bad cases the eye may be rendered almost completely immoveable by a glueing of both lids to it. Often, at the same time, the cornea is rendered opaque by the accident causing the symblepharon.

Sometimes the adhesion takes place between the lid and the cornea, and causes a still greater interference with vision than would have resulted from injury to the cornea alone had this not occurred. Adhesion at the inner and outer sides, by interfering with the lateral movements of the eye, may give rise to diplopia.

The operations which can be performed for these different cases are described in another Chapter.

DISEASES OF THE LACHRYMAL APPARATUS.

AFFECTIONS OF THE LACHRYMAL GLAND.—Inflammation of the lachrymal gland is a rather rare affection, which occurs both in an acute and chronic form. The acute inflammation is accompanied by very considerable swelling of the lid and conjunctiva, especially their external portions. The swollen lid is tender to pressure, and through the skin, which is moveable over it, can be felt the hard margin of the enlarged gland. Not only does the increased size of the gland cause considerable drooping and more or less complete immobility of the upper lid, but it may also give rise to some protrusion and depression of the eye itself. If it is possible to evert the lid, the lower portion of the swollen gland may be seen pressing forward the fold of conjunctiva forming the transition between the portions covering the eye and the lid. When an abscess forms, it may burst either through the skin or through the conjunctiva; in either case a fistulous opening is likely to result. A spreading of the purulent inflammation to the cellular tissue of the orbit has only been observed in a few cases.

The *treatment* should first be directed towards preventing suppuration by means of ice-compresses, counter-irritation with iodine, &c.; but as soon as there is fluctuation an opening should be made, preferably, if possible, through the conjunctiva as a fistula in that situation is not so awkward as one opening through the skin.

Inflammation of the lachrymal gland sometimes becomes chronic, or may be chronic from the first. Some of these cases are bilateral, and appear to be mostly syphilitic in their nature. The acute inflammation is most frequent in children, and the cause is usually some trauma. In chronic cases which resist any other treatment, the palpebral portion of the gland may be excised.

Simple Hypertrophy or Adenoma of the Lachrymal Gland is also met with, and sometimes the gland, though apparently not very much enlarged, sinks down into the upper lid, causing ptosis. In either case excision is necessary, care being taken not to interfere with the tendon of the levator palpebræ. Fibroma of the lachrymal gland also occurs.

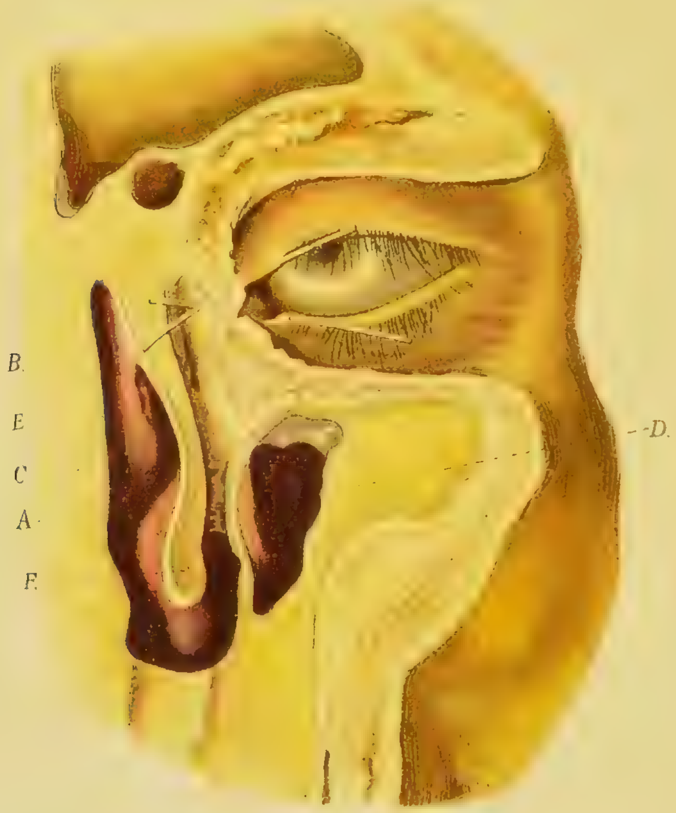


Fig. 19.—Transverse section of face from above downwards and slightly backwards from frontal sinus to first molar tooth. It opened nasal cavity (*A*), lachrymal sac (*B*), nasal duct (*C*), and antrum of Highmore (*D*).

Bristles were passed through puncta and canaliculi into lachrymal sac. Middle turbinated process (*E*). Inferior turbinated process (*F*).—From a preparation by Dr. Symington.

Fistula of the Lachrymal Gland arises from the bursting of an abscess of the gland, and has also been recorded as a congenital condition by Steinheim. Generally the fistula is an extremely narrow capillary channel, so that the tears which find their way through it may be little more than fine beads, which evaporate without flowing over the skin of the lid. There is then a tendency for it to close from time to time, and reopen after this has led to some little irritation from surrounding infiltration.

Treatment.—Cases which cause little or no trouble may be left alone. When the skin is much irritated by the tears, an attempt should be made to cure the fistula by means of a wire heated by the galvanic current, or by Bowman's operation, for diverting the opening from the outer to the conjunctival surface. Often any attempt to cure the fistula is unsuccessful, and recourse has then to be taken to partial excision of the gland.

MALIGNANT TUMOURS.—Both sarcoma and carcinoma occasionally affect the lachrymal gland. The diagnosis is usually not easy, as there is at first no difference between them and the simpler forms of hypertrophy. On this account it is better to make a practice of excising the gland in all cases where it is enlarged on one side alone, and the cause is not an acute inflammation. The removal of the gland does not interfere with the necessary supply of moisture to the eye, as, although the tear supply is thus removed, other secretions from the lids suffice for the purpose.

DACRYOPS, or a clear cyst due to distention of one of the lachrymal ducts with tears, is a rare affection of the lower surface of the upper lid. In one such case which came under my treatment, the cyst entirely disappeared after one puncture, but it is generally recommended to keep the opening made, patent for some time, as there is a tendency to reaccumulation.

DISEASES OF THE TEAR PASSAGES.—Affections of the parts connected with the excretion of the tears—(see Figs. 19 and 20) —are of frequent occurrence at all ages, and are consequently of considerably more practical importance than those which involve the secretory mechanism.

The proper removal of the tears down into the nose may be impeded by abnormalities in the structure and position of the canaliculi, by acute or chronic conditions of inflammation of the lachrymal sac, or by strictures in some parts of the lachrymal canal.

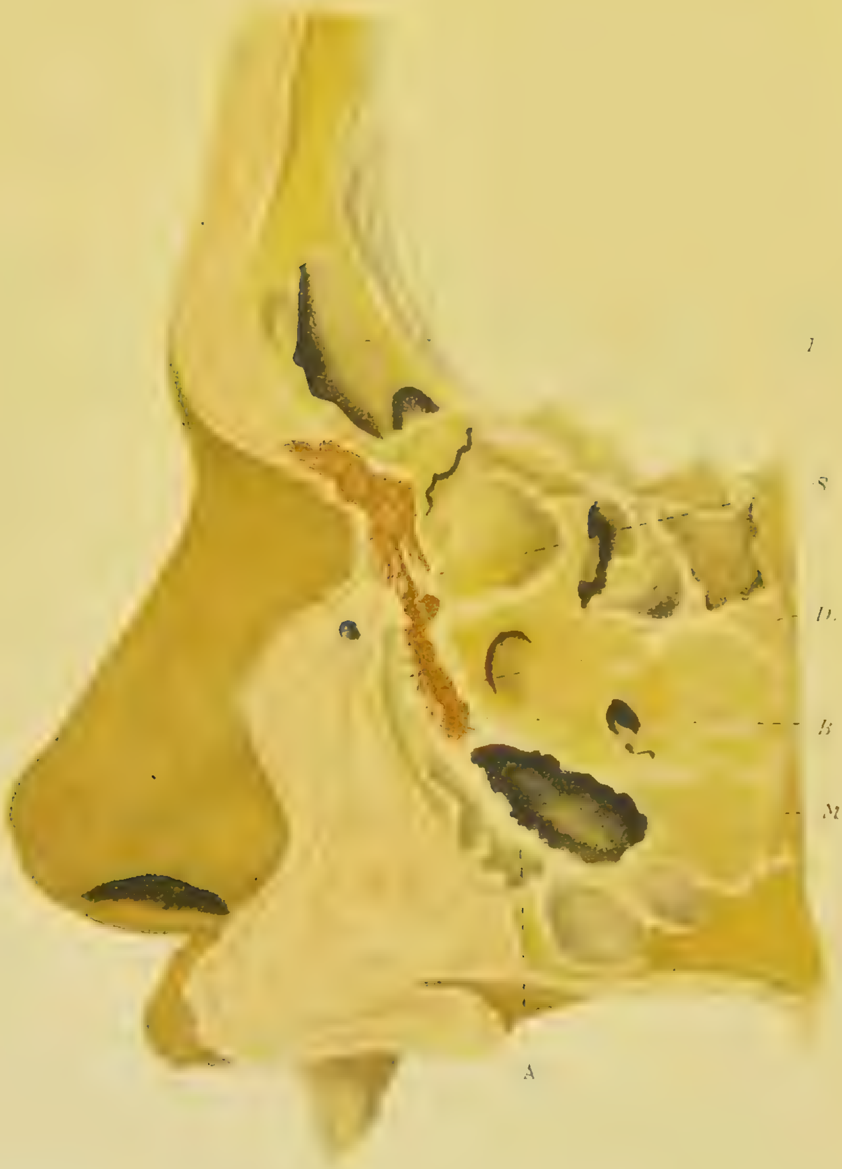


Fig. 20.—Oblique section made from above downwards, backwards, and slightly outwards in the direction of the tear duct. The section opened the lachrymal sac near its outer wall, and the lachrymal duct slightly towards its inner wall. The termination of the duct lies in the same sagittal plane as the outer surface of the ala nasi, and about one inch behind its junction with the cheek. It opens into the outer wall of the inferior meatus.

F, frontal sinus *M*, inferior meatus ; *S*, tear sac ; *D*, tear duct ; *B*, anterior wall of superior maxilla, forming anterior wall of duct ; *A*, antrum.—From a preparation by Dr. Symington.

Abnormalities in connection with the canaliculi, more especially of the lower ones, on which, under normal circumstances, most of the work devolves, are sometimes congenital, but more frequently acquired. Thus, cases are met with in which the puncta lachrymalia and canaliculi are absent or obliterated. Sometimes there are found supernumary puncta in connection with other, usually incomplete, canaliculi. A narrowing of the canaliculi leading to *epiphora*, or watering of the eye, is a frequent result of conjunctivitis. In the normal state the puncta are not seen without slightly everting the lids—they are applied to the eye. The tears find their way by capillarity between the lids and the eye to the puncta, being prevented from overflowing when not too copious by the oily margin of the lower lid. A slight eversion of the puncta, however, without any manifest ectropion, is sufficient to cause epiphora.

Where the condition of eversion is permanent, and not due to some temporary swelling of the conjunctiva, all that is necessary, as a rule, to cure the watery condition of the eye is to convert the canaliculus into an open rill. This method of treatment, first introduced by Bowman, is described, as it is now performed, with the narrow probe-pointed knife of Weber, in Chapter XVIII. The tears pass more readily into the open channel, and from thence into the tear sac and duct, so that after the slight irritation caused by the cut the overflow ceases. The same treatment is also generally the most applicable where the canaliculus is narrowed, but an attempt may sometimes be made to dilate it, by passing small probes along it. Electrolysis has been used for this purpose by Jessop and Steavenson.

Wounds of the lid, if they involve the canaliculus, are very apt to lead to troublesome epiphora after cicatrisation. When the patient is seen soon after the accident, care should be taken, if the edges of the wound have to be brought together, to avoid obliteration of the channel. The simplest way of doing this is to slit up the two portions of the divided canaliculus, and prevent them reclosing by occasionally running a probe along the rill thus formed, until the lid wound has healed. The attempt is sometimes made to maintain the canaliculus entire by leaving a piece of silver wire in, but this is very apt to be unsuccessful, and the result is in any case no more satisfactory, and much more troublesome to attain, than that which follows the simple slitting. In cases where obliteration has taken place just at the lower punctum—a rare condition, and generally the result of some localised inflammation or slight wound—the upper canaliculus will have to be slit, but if the punctum there be also obliterated, an opening may be got by cutting into the sac, passing a narrow probe along the canaliculus, and then cutting down upon it.

Sometimes foreign bodies get into the canaliculi, and more or less completely block up the passage for the tears. Occasionally one sees a portion of an eyelash sticking in, and this may, in rare instances, form the nucleus for an incrustation. Concretions that form in this situation have been shown to be mainly fungoid, being made up of masses of leptothrix.

POLYPI, or rather PAPILLOMATA, occur as a rare disease of the canaliculus. If they show much tendency to grow and cause epiphora, they should be removed. The affection is rather troublesome, and it is not easy to completely eradicate the growths without causing more mutilation than is compatible with the function of the parts.

INFLAMMATION OF THE TEAR SAC.—The mucous membrane of the tear sac is liable to become inflamed, and this gives rise to either an increase in the normal secretions or to the secretion of abnormal products.

Inflammation of the sac is known as *dacryocystitis*, and may be of different degrees of acuteness. As the mucous membrane of the sac swells, its lower orifice or passage into the tear duct becomes narrowed or closed, partly by the approximation of the swollen folds of the membrane which are met with in this situation, and partly, no doubt, by the increased mucoid secretion from the swollen surface. The result, in any case, is that the tears do not get properly down into the nose, and by their retention set up further irritation, while at the same time they flow over on to the cheek. The secretion, if the inflammation be merely catarrhal, may be mainly or wholly mucoid, and gradually, as it collects, it leads to distention of the sac. This causes a swelling at the inner angle of the eye, which gives a more or less elastic impression to the finger. Often by pressing firmly on this swelling the mucus can be pressed down into the nose. Such a condition has received different names, but it is most commonly termed a *mucocele.* The mucus may be perfectly clear and transparent, or more or less turbid from the admixture of pus cells.

The most common form of inflammation of the sac is a chronic blenorrhœa, in which, along with epiphora, there is no marked distention of the cavity, but in which, on pressure over the sac, some thin purulent matter escapes as a rule by the canaliculi. In a certain proportion of such cases, and relatively much more frequently in children than adults, the purulent inflammation becomes acute, and may lead not only to accumulation of pus in the cavity, but to inflammation of the superjacent tissues.

Acute Purulent Dacryocystitis generally causes œdema of the eyelids, which, if considerable in amount, may altogether mask the local swelling, so that any one unaccustomed to see such cases might well imagine that the lids or the periosteum round the border of the orbit was the site of the inflammation.

The *diagnosis* can readily be made by determining, on pressure with the finger, the point of greatest tenderness. In dacryocystitis no pain is felt until pressure is made over the sac, where even the slightest pressure is often exceedingly painful.

In some cases of acute inflammation of the sac, healing takes place without any bursting of the abscess; but in many the skin over the sac becomes more and more thinned, and eventually gives way. This usually takes place at a somewhat lower level than the sac itself after the tissues above have been infiltrated —at the most dependent part, therefore, of the superficial abscess thus formed. The result of this is that there is a passage of some length formed between the external and internal openings, which has a tendency to become fistulous. Sometimes a more or less extensive necrosis of the skin takes place, so that an ulcer is established in this situation, which may lead to cicatricial ectropion of the lower lid.

Dacryocystitis is most frequently set up by an inflammation originating in the mucous membrane itself, and is probably as a rule the result of extension of a similar condition from the mucous membrane of the nose, only increased in severity by the irritation which the retention of the secretion produces. Sometimes it appears to be primarily associated with disease of the bone or periosteum, which is then of a strumous or syphilitic nature. The ordinary inflammation beginning in the sac rarely, if ever, spreads to the bone; so that in cases where the bone is found to be diseased, and not merely bare, as may result from rough treatment, such disease must be looked upon as the primary affection.

A correct idea of the etiology of inflammation of the lachrymal sac is of importance in suggesting a suitable plan of treatment. There can be little doubt that by far the largest majority of cases begin as a cold in the head, an affection from which few escape altogether, but to which some are much more liable than others. If the swelling of the mucous membrane of the lachrymal duct persists for long, there is a tendency, when any individual predisposition exists, to the stagnation and decomposition of the secretions in the sac, which becomes more and more pronounced the more its anterior wall becomes distended. This sets up an irritation, and leads, as a rule, to blenorrhœa of the sac, which may be more or less pronounced, and which is liable at any time to pass into the acute form of dacryocystitis. When this takes place there has generally been a fresh coryza shortly before; or, if this is not the case, some increased swelling in the mucous membrane of the duct, or some increased virulence of the decomposing secretion lodged in the sac, must be looked upon as the cause of the symptoms becoming more severe. It is remarkable how seldom any complication in the sac is met with in cases of purulent conjunctivitis. I have occasionally seen dacryocystitis after ophthalmia neonatorum, but only in cases where there have been more or less marked, probably syphilitic, changes about the mouth and nose, to which the condition might more readily be ascribed. It is conspicuously absent after gonorrhœal conjunctivitis. These facts render it certain that a spreading of a conjunctival affection to the mucous membrane of the tear sac is at all events only a very exceptional cause of blenorrhœa of the sac. Hansen Grut goes as far as to say that "affections of the conjunctiva, met with along with blenorrhœa of the sac, are either accidental complications or are caused by it."

The secretion which passes into the conjunctival sac from the abnormally inflamed tear sac is not infectious, and never leads either to purulent inflammation of the conjunctiva or to conjunctivitis if transferred to the other eye, though it is more or less irritating, and thus gives rise to blepharitis. It possesses, therefore, none of the virulence of the pus which is found in a case of gonorrhœal conjunctivitis. Nevertheless, it is well known to be a source of great danger when any operation is performed on the eye, and the cause in many cases of the septic hypopyon ulcer which follows slight abrasions of the cornea.

By many, strictures of the duct are looked upon as the immediate exciting cause of dacryocystitis; yet it is remarkable in what a large proportion of cases probes can be passed with the greatest ease when once the sac has been opened into. Many cases of stricture, where the stricture is far down at the entrance of the duct into the nose, do not give rise to inflammation of the sac, and as for the strictures which are met with in the other situations where they are common, viz., at the lower orifice of the sac, these, as Hansen Grut has pointed out, are almost invariably the consequence of the destructive inflammation of an acute dacryocystitis, and not its cause.

The *treatment* of blenorrhœa of the tear sac should aim at preventing the occurrence of acute phlegmonous inflammation; and stopping as far as possible the interference with the discharge of the tears. The first, however, is the most important; but as many cases are first seen when the stage of acute inflammation has already begun, it is also necessary to consider the proper course of treatment to be adopted under such circumstances.

As soon as the existence of a chronic blenorrhœa has been diagnosed, active treatment should be begun without delay. This should consist in making an opening into the sac from above, through one or both of the canaliculi, probing the duct from time to time, so as to maintain its patency, and by astringent and antiseptic injections modifying, as far as possible, the character of the secretion from the mucous surfaces.

The choice of which canaliculus to open is perhaps hardly a very important matter, still there are good reasons for preferring the upper. In the first place, the lower is then left in its physiological state, and as it has most to do with the removal of the tears, this may be some, though possibly only a very slight, advantage. A more practical consideration is the circumstance that the upper canaliculus is in a more direct line with the duct, so that probes can be passed without stretching the tissues at the opening into the sac to the same extent as is often done when the lower canaliculus is opened, and which may lead to cicatricial contractions in this situation, and consequently to a complication which is apt to prove troublesome.

The canaliculus should be freely opened into the sac in the manner described in the chapter on operations, and the knife then passed into the sac itself, with the cutting edge directed forward, and then made, by a slight sawing movement and slight rotation on its axis to either side, to freely divide any constricting bands which are present in this situation. After this has been done, a medium-sized probe should be passed down the duct into the nose. One of the most convenient kind of probes are those introduced by Argyll Robertson, which are made of soft silver, and can thus easily be bent to any shape required. As has been already said, the probe can usually be passed without the slightest difficulty. It may be left in for five or ten minutes, and when withdrawn, the patient is directed, while shutting his mouth and holding his nose, to blow so as to puff out the cheeks. This sends a stream of air up the duct which carries with it some of the secretions and shows that it is free. In the normal state, owing to the valvular nature of the opening at the lower end of the duct, and the narrowness of the canaliculi, it is rarely possible to blow air through in this way, and soon after probing, too, the attempt is usually unsuccessful. The same probe may subsequently be passed again at intervals of two or three days, and then followed by an injection of carbolic acid (1-40) through the duct.

There is no necessity for passing larger probes, as is often done, in the belief that the most important part in the treatment consists in maximally dilating the duct. I have tried such probes, especially those introduced by Couper, and am convinced that they either do no good, or are positively hurtful. The injections are best made with a hollow probe, which is first passed into the nose, and then slowly withdrawn as the fluid is forced through. In this way every part is thoroughly cleansed. By this treatment a great improvement usually takes place in the course of two or three weeks, but the length of time during which the treatment has to be continued depends a good deal on the chronicity of the case. The reason of this seems to be the tendency to greater and greater distention of the sac, and consequently a greater likelihood of stagnation taking place in it the longer the case has continued.

In the case of acute dacryocystitis, if seen before the skin over the sac has become very much thinned, a free opening should be made into it by slitting both canaliculi and pressing out the pus. The inflammation will then, as a rule, subside, and after a few days admit of probing and syringing. In such cases it is not advisable to probe at once, not only because the state of acute inflammation makes it very much more painful, but also because the swollen and softened condition of the mucous membrane renders the probing difficult without producing lacerations, the contractions following which are liable to give rise to further mischief. Where the overlying tissues have already become purulently infiltrated when the case first comes under observation, the sac must be opened into by a vertical incision. The opening should be free, and not be allowed to close for some days, until the discharge has much diminished and the swelling gone down. The wound may be dressed with cotton wool dipped in corrosive sublimate and covered with gutta-percha tissue. Afterwards the upper canaliculus should be slit, and the duct probed and syringed in the manner already described. Occasionally there may be left after this treatment a fistulous opening into the sac. If this should remain permanent, it can be obliterated by passing a red hot wire along it. A fistula is much more likely to remain when spontaneous bursting of the abscess is allowed to take place.

The treatment in cases which have gone on to acute dacryocystitis is often much more difficult and unsatisfactory, on account of the great distention of the sac which has taken place, and also not infrequently owing to the narrowing of the orifice between the sac and the duct, which necessitates more frequent probing. When a stricture exists in this situation, the patient may, after it has been dilated, be taught to pass the probe himself, or, as has been suggested and practised by Benson, he may introduce pewter styles every night, removing them again in the morning. By prolonged treatment of this nature more or less improvement is generally eventually obtained, but there are always some cases in which this is not the case. In such it is necessary to have recourse to destruction of the sac altogether. This may either be done by freely cauterising its mucous surface with the thermo-cautery, or by extirpating it altogether The latter is, on the whole, the most satisfactory proceeding. Curiously enough, although the result of destroying the tear sac is to interfere completely with the excretion of the tears, it is rare that any uncomfortable degree of epiphora afterwards remains; a fact which points very strongly to the main cause of the troublesome epiphora, in most cases of blenorrhœa of the sac, being due to other conditions than the interference with the normal patency of the duct; and which explains why so many cases improve as soon as the nature of the secretions has been modified by antiseptic applications.

Stricture of the Lachrymal Duct.—The diagnosis of stricture of the duct cannot be made except by probing. It may or may not exist along with blenorrhœa of the sac. The stricture, except in cases where there is disease of the bony canal, occurs mainly in two places—at the opening of the sac into the duct, and at the nasal termination of the duct. Cases of true stricture, and not mere swelling of the mucous membrane alone, are usually very troublesome. An attempt may always be made to dilate the stricture by the introduction of the probes at intervals of a few days, or by causing the patient to wear styles, but the results are often unsatisfactory; fortunately, however, such cases are comparatively rare.

As has been already said, the affections of the lachrymal sac and duct are of very great practical importance, not only on account of their frequency, but also because of the dangers to which the eye is thereby exposed. A very large proportion of cases of one-sided blindness are cases of dense leucomata, following hypopyon ulcer of the cornea; and that, again, is in a large percentage of cases the result of infection from a blenorrhœa of the sac. A very considerable proportion, too, of the cases which "go wrong" after operations have the same infectious cause.

As to the frequency of lachrymal disease, I find 4604 cases recorded out 255,730 patients treated during four or five years in some of the principal English hospitals,—that is, a percentage of 1·88. Comparing the numbers for different years, I find that they vary from less than 1·3 per cent. to more than 2·5 per cent., a degree of variation which, as the numbers taken are large, can hardly be considered as wholly accidental, but points to the existence of climatic influences, and suggests the causal connection between coryza and lachrymal affections referred to above.

CHAPTER III.

DISEASES OF THE CONJUNCTIVA.

HYPERÆMIA OF THE CONJUNCTIVA.—The vessels of the conjunctiva become abnormally injected as the first stage in inflammation of that membrane. Hyperæmia of the conjunctiva may, however, exist without passing into inflammation. There is then no marked increase in the mucoid secretions, although always more or less lachrymation. The vessels of the palpebral conjunctiva are more injected than those of the ocular conjunctiva, which in many cases do not show any abnormality in this respect. The conjunctival fold, which is usually pale, is injected. The eyes feel hot and heavy, and are easily tired by any attempts at reading, or by any exposure to strong light. Great differences are met with in the severity of these symptoms. Some individuals are liable to recurrent attacks of acute hyperæmia, which suggests the presence of some vasomotor disturbance. In others, a chronic condition of hyperæmia remains for a long time, rendering them unfit for any prolonged use of the eyes. Hyperæmia of the conjunctiva is set up by local irritation, and by various states of general health, more especially disorders of the liver.

The *treatment* should consist in attention to the general health—avoidance of close, smoky atmosphere, and the occasional sponging of the eyes with very hot water. At the first examination the lids should be everted, so that anything which might possibly be lying in the conjunctival sac may be removed; and for the same reason it is well to use three or four times a day a solution of boracic acid (1 in 50) to bathe the eyes with. Astringent lotions should, as a rule, be avoided. Sometimes it is necessary to protect the eyes from light by means of darkened glasses, but this should not be encouraged for any length of time, as it is extremely liable to increase the sensibility.

CONJUNCTIVITIS.—All the different forms of inflammation, catarrhal, purulent, and membranous, which occur in other mucous membranes are met with in the conjunctiva.

Two other forms, rarely occurring elsewhere, are also common, viz., granular and phlyctenular conjunctivitis.

SIMPLE OR CATARRHAL CONJUNCTIVITIS is a common affection, characterised by hyperæmia and swelling of the conjunctiva, with increase of the secretions. This gives rise to pain and heat in the eyes, as well as to a feeling as if there were constantly some foreign body, dust or sand, in them. The lids feel heavy to raise, and in the morning, or after having been closed for some time, are more or less glued together by the secretions which have dried on the eyelashes. The hyperæmia, when intense, may give rise to a serous thickening, which is most marked at the conjunctival fold; and, at the same time, in many cases little papillary elevations are met with in the same situation. In very severe cases the œdema extends to the ocular conjunctiva, a condition to which the name *chemosis* is given. It may even extend, in a slight degree, to the lid as well. Not infrequently small ecchymoses also occur in the conjunctiva.

Catarrhal conjunctivitis is almost always bilateral, one eye being often affected a few days after the other. It occurs most frequently at certain times of the year, more especially spring and autumn. It is met with either in an acute or in a chronic form. The chronic is frequently preceded by an acute attack. In the chronic condition the injection is mostly or entirely confined to the conjunctiva of the lids, which at the same time presents a more or less velvety appearance, owing to the presence of numerous injected papilliform elevations. An eczematous thickening and excoriation of the skin of the lower lid, particularly at the outer angle of the eye, is often met with at the same time, and in long continued cases there may be as well a certain degree of eversion of the lower punctum lachrymale. The interference with the absorption or outflow of the tears, to which this condition gives rise, tends to keep up the inflammation. The irritation of the margin of the lids, also, often leads to a complication with blepharitis.

Acute catarrhal conjunctivitis is rarely complicated by any corneal inflammation, and does not cause any permanent defect of vision. Besides the constant blinking and photophobia, from which patients with this affection suffer, they often complain of temporary obscuration of vision, or of seeing haloes or rays round lights, or sometimes of polyopia. All these appearances are due to little pieces of secretion in front of the cornea, which produce alterations in the refraction of the rays of light entering the eye. They disappear on rubbing the eye, but in many cases quickly reappear.

The secretion from a catarrhal conjunctivitis is certainly contagious, if the affection be acute, when it is muco-purulent in character. In the more chronic forms there are probably considerable individual differences in this respect; but, on the whole, the discharge in such cases is not by any means markedly contagious.

Conjunctivitis may be due to the spreading of a catarrhal inflammation from the mucous membrane of the nose and throat, or may more frequently be set up independently by any mechanical, thermal, or chemical irritation, caused by the presence of dust, smoke, coal, vitiated atmosphere, or any foreign substance remaining for some time in the conjunctival sac. When chronic, and on the one side only, there is almost certainly some connection with inflammation of the lachrymal sac. An acute attack of catarrhal conjunctivitis does not usually last for more than ten days or a fortnight.

In the *treatment* of conjunctivitis attention should first be directed to the cause. The lids should be everted to discover if any foreign bodies are lodged in the conjunctival sac, and the condition of the tear sac and canal examined into. Any unusual irritation by dust, smoke, or sand, or by strong light, should be as far as possible avoided. The local treatment should consist in the frequent bathing of the eyes with some weak non-irritating antiseptic lotion, such as boracic acid (1-50) or corrosive sublimate (1-5000 or 1-10,000). Care should be taken that all the secretion which has dried on the eyelashes is removed. When there is much discharge, nitrate of silver in solution (5-10 grains to ℥i.) should be applied to the everted lids once daily or every second day. In applying this caustic solution the object aimed at is to form a protecting eschar of the albuminate of silver on the surface of the conjunctiva, and therefore care should be taken that the conjunctival surface is dry before the solution comes in contact with it, otherwise it is at once precipitated by the tears as the chloride of silver. In order to avoid irritating the cornea, the excess should be neutralised with water. The best plan is, therefore, to dry the everted conjunctiva with a clean rag, then to apply the caustic solution with a small camel's-hair brush, and, after waiting ten or twelve seconds, to dip the brush in water and brush freely over the surface which has been painted.

No other astringents should be used during the acute stage, as they only tend to keep up the inflammation and make it chronic. Poulticing or fomenting, which is frequently resorted to, must also be avoided. A little simple ointment may be smeared along the edges of the lids at night to prevent them being glued together, and the discharge consequently retained.

When the conjunctivitis has become chronic, the treatment should depend greatly upon the appearance presented. If the conjunctiva is much swollen and hyperæmic, and there is at the same time a considerable amount of discharge, the nitrate of silver should be used as in the acute form, along with antiseptic applications. In many cases, too, lead lotion (2 grains to ℥i. of the neutral acetate) does good. Other cases, in which there is not so much swelling and discharge, are benefited by some astringent application,—solution of alum, tannic acid, sulphate of zinc and copper, &c. Of these, alum in the strength of from 2 to 8 grains to ℥i. is perhaps the most useful. Tannic acid may be used as a wash in the strength of 2 grains to ℥i., and with the addition of ʒi. of glycerine to each ℥i. of water. In cases where a good deal of swelling of the conjunctiva exists, principally of the fold, without much hyperæmia, and with more or less glutinous secretion, nitric acid (1 in 1000) may be tried in addition to the astringents mentioned. Sometimes it is necessary, in order to get rid of a chronic conjunctivitis, to wear darkened glasses, but this treatment should not be resorted to too much, as it often has not only little influence on the inflammation, but is apt to give rise to more or less hyper-sensitiveness of the retina. Finally, in all chronic cases attention must be paid to the general health, and any existing digestive disorders, or any gouty or rheumatic tendency, counteracted as far as possible.

Spring Catarrh.—A peculiar variety of recurrent chronic conjunctivitis has received the name of spring catarrh. This is a rather rare affection, met with in young individuals, and characterised by an injection and swelling, limited to the pericorneal portion of the ocular conjunctiva. The elevations which are met with in this situation, and which either extend all round the cornea, or are limited to some portions of its margin, are solid pinkish or greyish milky-looking masses, which show no tendency to ulcerate. In this respect, as well as on account of their solidity, they are readily distinguished from phlyctenules which they at first sight sometimes resemble. There is, besides, associated with this affection, a more or less characteristic milkish-white coloration of the conjunctiva as a whole. In some cases the condition is accompanied by some increase in the secretions, but rarely to any great extent. Slight infiltration of the corneal margin may occur—never, however, extending far in; and, beyond a sometimes high degree of photophobia, the affection does not give rise to any symptoms of consequence. Not unfrequently flattened masses of cartilaginous-looking granulation tissue make their appearance on the surface of the tarsal portion of the conjunctiva of the upper lid. These may attain an enormous size, and such cases are liable to be mistaken for trachoma. The inflammation has, even more than other forms of conjunctivitis, a tendency to recur every spring, and to continue, when once set up, for several months. In many cases the improvement which takes place at the beginning of the winter appears not to go on to complete recovery, so that the condition in spring may be looked upon more as an exacerbation than as a true recurrence. Spring catarrh is in reality a hyperplasia of the epithelium alone; there is no deep infiltration.

Treatment.—Astringent lotions should be avoided. If there is much photophobia, dark glasses may be used. Excision of the granulations when they are massive, or, still better, burning them down carefully with the thermo-cautery, is the best treatment. No cicatrix need be left if this is properly done.

PURULENT CONJUNCTIVITIS.—Although an acute attack of simple conjunctivitis is often accompanied by considerable purulent discharge, it is nevertheless important, from a clinical point of view, to make a distinction between this and the more characteristically purulent forms of inflammation, viz., *gonorrhœal conjunctivitis* and the conjunctivitis of new-born infants, or what is generally known by the name of *ophthalmia neonatorum*.

Gonorrhœal Conjunctivitis, or gonorrhœal ophthalmia, is one of the most serious inflammations met with in the eye. It is set up usually by the direct transference of pus from a gonorrhœa into the conjunctival sac. Such pus is now known to acquire its virulence from the presence of characteristic micro-organisms, to which the name of *gonococci* is given. The first symptoms begin several hours, or it may be a day or two, after inoculation. The appearance during the first day may be such as to suggest merely an acute attack of simple conjunctivitis. More frequently the rapidity with which the symptoms set in, and especially the early appearance of more or less chemosis, excites suspicion, and leads to the detection of the source of inflammation. The pus finds its way to the eye from a urethral or vaginal gonorrhœa, or from a similar inflammation in some other eye, so that the inoculation may either be from the individual himself, as is most commonly the case, particularly in young men, or from some one else whose towel has been soiled by the discharge, and used subsequently by the patient. The great importance of warning patients suffering from gonorrhœa, (and, in the case of women, from any other discharge as well), of the danger to their own eyes, and those of others living in the same room, of using towels, sponges, &c., in common, and of the consequent necessity of absolute cleanliness, cannot be exaggerated.

Considerable differences exist in the length of time before the inflammation reaches its full height and leads to a copious purulent discharge. What is usually distinguished as the first stage, in which only a watery and slightly mucoid discharge, often considerable in amount, takes place, may last a couple of days or more, or may rapidly pass on to chemosis, due to a fibrino-plastic exudation beneath the conjunctiva, and to swelling and redness of the lid. The second stage, which is characterised by the purulent discharge in addition to the chemosis and swelling of the lid, may then be reached on the second day after the first symptoms have made their appearance, or may be delayed, and this is more common, till the third or fourth day, or even later.

The difference in the rapidity of development has been shown to stand in close connection with the amount of active inoculating matter which enters the eye. Small amount, dilution, and dessication all retard the onset as well as the rapidity with which the abnormal changes succeed each other. Yet the resulting inflammation may be, and most frequently is, just as violent as that which results from inoculation with a more copious, and, at the time, a more virulent secretion. This fact would of itself point to the connection of micro-organisms with the affection, even if this were not definitely established. It is also of practical importance, in so far as it indicates the possibility of checking the disease in some cases by timely antiseptic measures.

Besides the appearances described as characterising the first stage of gonorrhœal ophthalmia, more or less pronounced subjective symptoms—pain, burning and neuralgic in character, photophobia, and lachrymation—are constant, and increase as the tenseness of the swelling of the conjunctiva and lid becomes greater and greater. The swelling of the upper lid generally reaches such a degree as to cause it to hang down over the lower lid, and to assume a red glossy appearance on its surface. It is then perfectly impossible for the patient to open his eye, or even for it to be raised to any extent with the finger. The full height of the inflammation is generally sustained for from one to three days, after which the skin of the lids regains some of its natural wrinkling, and the circulation in the conjunctiva becomes freer. The discharge continues as copious as before, but less flocculent, and the conjunctiva becomes redder and softer.

The hardness, caused by the serous and often fibrino-plastic sub-conjunctival effusion during the course of the inflammation, is a source of danger to the cornea, whose nutrition, derived as it is from the surrounding vessels, becomes thereby more or less interfered with. It is not uncommon to find, therefore, a steaminess or loss of transparency of the cornea, which at the time of examination may or may not be associated with ulceration. This ulceration is often near the margin of the cornea, and is then hidden by overlapping portions of chemosed conjunctiva; it has a tendency to extend, not only in depth, but also circumferentially. When extension takes place in the latter direction, the result is apt to be a necrosis of the whole central portion of the cornea, and a consequent hopeless destruction of vision. A complication with corneal ulceration may also be met with at a later stage of the disease, during the period of retrogression. The ulcers then formed have not unlikely quite a different etiology and are more amenable to treatment, and consequently less likely to lead to total destruction of the cornea. They are usually, though such an origin cannot always be traced, due to the inoculation of the corneal tissue with the pus from the conjunctiva. As long as the conjunctival discharge continues there is the possibility of a complication of this nature, although the danger diminishes every day. On this account it is important to avoid any rough handling during the treatment of the lids, as any abrasion of the cornea very much increases the risk of inoculation. Most likely corneal affections met with at any stage of the conjunctival inflammation are, properly speaking, infective. While, however, the later ones are only liable to make their appearance where the cornea is abraded, the more serious early ulcerations may be accounted for by supposing that there is first an interference with the vitality of the superficial layers, and subsequently inoculation.

In acute cases, complete recovery of the conjunctival inflammation, and a return to the normal condition without any loss of substance, takes place in from three to six weeks. Some cases gradually lapse into a chronic state. In these cases the conjunctiva often remains enormously swollen for months, the tarsal portion assuming a thick and more or less uniform velvety appearance, while the retrotarsal folds present large fleshy elevations and furrows, which protrude into prominences when the lids are everted. Not unfrequently this leads to *ectropion.* The lower lid is more often everted, on account of the smaller size of the tarsus, and the thickening of the skin produced by excoriation of the overflowing tears and secretion. Occasionally, however, the upper lid is everted as well, the everted portion being often hardened and encrusted if not properly attended to.

While in all conditions the cornea is liable to ulceration from inoculation, the more serious, and, properly speaking, necrotic form is less liable to make its appearance, or at all events to show the same tendency to circumferential extension in cases where a previous abnormal vascularity has existed. This circumstance is taken advantage of in the treatment by inoculation of cases of dense superficial vascular keratitis or pannus.

In the *treatment* of gonorrhœal ophthalmia the first consideration, when only one eye is affected—and this fortunately is the rule—must be to protect the second from inoculation. This may always be done by tying it up in the following way:—The lids are gently closed, and kept so by two or three strips of court plaster. The hollow between the nose and eye is then filled up with cotton wool, preferably absorbent, or absorbent containing some antiseptic; over this a piece of gutta-percha tissue is placed, and the whole bound down along the edges with strips of closely fitting india-rubber plaster. Special care must be taken that this adheres well along the nose, and the patient should as much as possible, when in bed, lie on the side of the affected eye, to prevent the discharge trickling over to the other side and possibly loosening the plaster. If properly applied, there is, however, not much fear of this. Before tying up the sound eye it is well to thoroughly wash it out with an antiseptic solution, and for this purpose a solution of corrosive sublimate (1 in 4000) is very serviceable, and now-a-days usually at hand in most places. In this way the effect of inoculation, which may have taken place shortly before the patient seeks advice, may in some instances undoubtedly be neutralised.

It is certainly very trying for the patient to have the good eye occluded from vision at the time when the other, owing to the great swelling of the lid, is temporarily useless. He is thereby rendered very helpless and unable to go about alone, so that his spirits, generally depressed as it is, are not raised by his being put entirely in the dark. On this account it is well, where this is possible, to protect the sound eye in a manner which does not altogether interfere with its use. This can be done tolerably efficiently by means of a plaster which generally goes by the name of *Buller's shield* (see Fig. 21), and which consists of a watch-glass, kept in position in front of the eye by being held between two pieces of india-rubber plaster, in which a round aperture is cut, and the uppermost one of which extends to either side and above beyond the lower, so that the whole shield can be plastered down on to the nose, forehead, and cheek, and only left free below to allow of the circulation of air, and thus prevent the glass becoming obscure. This plan has the advantage of allowing one to observe the sound eye all along, and therefore the shield can be left on continually, whereas the ordinary occlusion bandage has frequently to be reapplied.

Having secured the sound eye, and taken precautions to prevent contagion in others, attention must be now directed to the inflamed one. The treatment to be adopted will depend on the stage of the inflammation and the state of the cornea. In the first stage, when the discharge is only serous and the chemosis slight, pretty frequent washings with corrosive sublimate solution (1 in 5000) and ice-compresses frequently changed may be used, and the patient's room, too, should be well ventilated and slightly darkened. When the inflammation comes to its acme the ice may be discontinued, and nothing used but a little vaseline smeared along the edges of the lids and the antiseptic

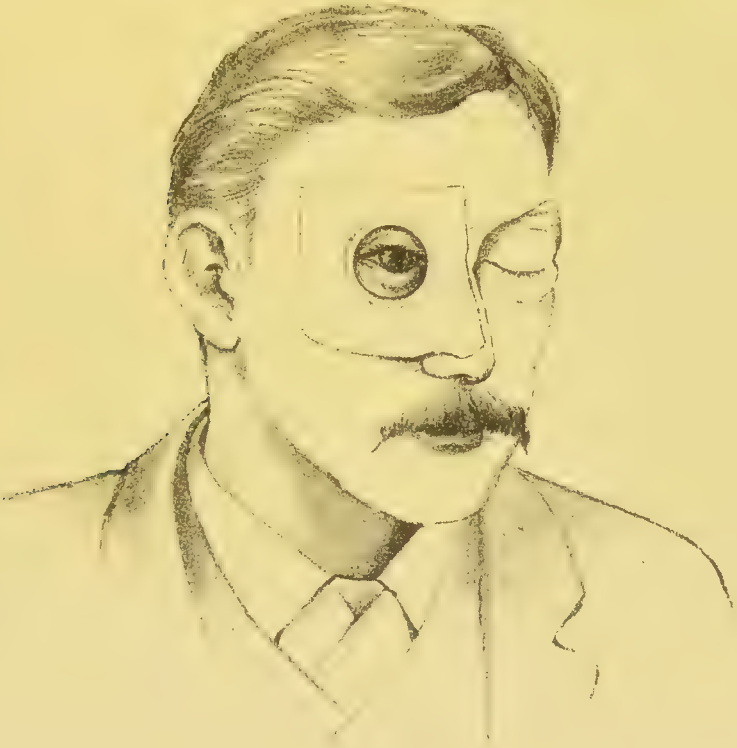


Fig. 21.—Buller's shield.

wash as before. Some recommend local bleeding. This is of doubtful use, but may be tried in strong healthy individuals. The readiest and perhaps the best plan to effect this is to make a good large cut across the outer canthus with a pair of scissors. This at the same time tends to relieve the tension of the swollen lid on the eye. It is rare that at this stage the upper lid can be everted without the greatest difficulty, so that any attempt at the local application of caustic solution, as is often recommended, is neither desirable, nor always indeed possible. The cornea must be watched, and any symptoms of loss of vitality counteracted, if possible, by free incisions made with scissors, and preferably in a radial direction, in the chemosed conjunctiva. Unfortunately this treatment is by no means always efficacious in arresting the dreaded complication, owing no doubt to the interference with nutrition being not only due to pressure on the anterior ciliary vessels, but to actual stasis in them. Hansen Grut practises excision of a portion of the swollen conjunctiva all round the cornea. When this treatment is adopted it should be done early. As soon as the upper lid can be everted without too great difficulty, an application of a solution of nitrate of silver (5–10 grs. to ℥i., not stronger) should be made once or twice during the twenty-four hours, with the object of restraining the discharge and diminishing the risk of subsequent inoculation of the cornea. Any ulceration of the cornea appearing at this stage may be treated by applying directly to it the same caustic solution, unless it has begun to heal, when care should be taken that the nitrate of silver does not come in contact with it. During the whole of the time free exercise in the open air is desirable. Attention should also be given to the action of the bowels, and opiates given, should sleep not be obtained without.

Ophthalmia Neonatorum is practically the same disease as that just described; that is to say, it is due to an inoculation with a specific virus. The virus is probably not always the same, or at all events what may be specifically the same, has suffered some modification, as there are greater differences met with in the intensity of this form of inflammation than can well be accounted for by merely individual receptive differences on the part of the patients.

Inoculation of the eyes of infants takes place in two ways, either directly from the maternal passages during birth, or afterwards by the use of soiled linen, sponges, &c. In the first case the inflammation usually begins on the third day, although it may be delayed two or three days longer, and both eyes, as a rule, are affected. In the second case the first appearances are often later in presenting themselves, and not so frequently in both eyes simultaneously. Purulent inflammation beginning after the first week is almost certainly due to some extraneous source of inoculation. The liability to purulent conjunctivitis in the new-born infant is greater the more prolonged the birth. Thus Mules found 80 per cent. of all cases in the children of primiparæ, and a larger number of cases amongst male than amongst female infants, owing, no doubt, as he suggests, to the fact that the head, being larger, is apt to rest longer on the perineum before being born.

Ophthalmia neonatorum is not by any means as a rule so severe an inflammation as purulent conjunctivitis in the adult. In the great majority of cases it is mainly the palpebral conjunctiva which is affected, so that the cornea does not run the same danger. The corneal complications which do arise are mostly the result of unskilful handling and want of cleanliness; that is to say, they are of the directly infected and not the necrotic type, and can often be arrested by proper treatment. It is rare indeed that we feel so helpless with respect to them as is the case with the early ulcerations in gonorrhœal ophthalmia.

With respect to the *treatment* in ophthalmia neonatorum, which should be on much the same lines as that for the other clinical form of purulent conjunctivitis, we have to consider as well the very important question of prophylaxis. That is to say, given the possibilities of inoculation taking place or having taken place during birth, how are the effects to be warded off? This question is of the utmost importance, because it has been shown that in many places the blindness resulting from this early inflammation constitutes about one-half of the total number of cases of blindness. Fortunately the results of systematic attempts made in this direction have been very successful, and in some larger lying-in hospitals brought about the almost complete disappearance of the disease. Irrigation of the vagina during labour with a solution of corrosive sublimate

(1 in 2000) in cases where a vaginal discharge exists, has been found to be a very successful prophylactic. The simplest, and at the same time efficient, treatment after birth is that of Credé, viz., to drop into the conjunctival sac a solution of nitrate of silver (10 grs. to ℥i.). In some cases this produces a little irritation, but it may quite safely be employed. Washing out with chlorine water or corrosive sublimate solution up to the strength of 1 in 2000 is also of use.

When the inflammation has actually begun, the local application of nitrate of silver (10 grs. to ℥i.) may be begun, and continued once daily as soon as the discharge becomes purulent, and this, along with frequent antiseptic washes and the application of a little fresh lard to the edges of the lids to prevent retention of the secretion, is all that is required. When only one eye is affected, the mother or nurse should be instructed to keep the child as much as possible from lying on the other side, so as to prevent the discharge trickling across the nose. When the discharge has almost ceased, the daily application once or twice of a solution of alum (4–8 grs. to ℥i.) may be substituted for the caustic solution. Most cases recover without entering upon what can properly be called a chronic state.

In a good many cases of ophthalmia neonatorum the conjunctival surfaces of the lids are found to be covered by a membrane. This form of *membranous conjunctivitis* must be distinguished from diphtheritic conjunctivitis. The membrane is easily detached, and although there is a tendency towards bleeding from the conjunctiva when it is rubbed off, it can readily be seen not to be due to any intimate adherence between the membrane and the conjunctiva, such as characterises diphtheritic inflammation. The abundance of the purulent secretion, as well as the absence of the livid grey coloration and marked constitutional disturbance, also distinguish this form from the much more uncommon diphtheritic conjunctivitis. The treatment is the same as for the ordinary ophthalmia neonatorum, with the exception that it is generally advisable not to use nitrate of silver so frequently nor so strong.

The discovery by Neisser in 1879 of a special micro-organism in the pus from cases of blenorrhœa neonatorum, as well as from gonorrhœal secretions, and to which he gave the name of *gonococcus*, led to a number of investigations by gynæcologists and ophthalmologists with the view of determining its morphological and pathogenetic characteristics. Neisser's views as to the specific nature of the gonococcus were confirmed by Haab and Sattler, though there were other ophthalmologists, as well as many gynæcologists, who opposed these views. One of the most important evidences in support of Neisser was given by Zweifel, who found that lochial secretions, in which no gonococci can be detected, can be introduced into the conjunctival sac of the newly-born child without fear of producing blenorrhœa. Kroner, having examined the secretions in ninety-two cases of ophthalmia neonatorum, with the result that in sixty-three cases he was able to detect gonococci, while in twenty-nine they appeared to be absent, came to the conclusion that two distinct forms of this ophthalmia exist.

The most complete and valuable contribution to this subject has been recently made by Bumm. Before the publication of his investigations there was an absence, as he himself remarks, "of any complete agreement, either as to the diagnostic importance of the gonococcus, from a clinical point of view, or its true relation to the diseased mucous membrane. The pathogenetic properties of these microorganisms had also not been established beyond all doubt." The following are the chief results of Bumm's investigations. The gonococcus is of a figure of 8 shape, *i.e.*, divided into two distinct parts, each of which is approximately hemispherical. It forms chain-like groups, owing to its increasing by fissure in all directions alternately at right angles to each other (see Fig. 22). Their average length is 1·25μ., but, according to the stage of their development, they are met with in sizes varying from 0·8μ. in length, and 0·6μ. in breadth, to 1·6μ. in length and 0·8μ. in breadth. The shape is not characteristic of the gonococci alone, there being other germs of the same genus (*Diplococcus*) which are innocuous, and cannot be distinguished from gonococci by the highest powers of the microscope. A special characteristic of the gonococcus is, however, the arrangement of them in roundish masses within the protoplasm and surrounding the nuclei of living cells. In cases which have not been treated with disinfectants they can always be demonstrated in the secretion from a gonorrhœically inflamed mucous membrane. Secretions free from gonococci produce no infection in mucous membranes, whereas, even in small quantities, a secretion containing gonococci will produce with absolute certainty a blenorrhœic inflammation of any mucous mem-

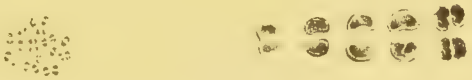


FIG. 22.—Gonococci (after Bumm).

brane with which it may be brought in contact. Examinations of twenty-six different conjunctivæ, from all stages of inflammation between thirty-six hours and the thirty-second day after birth, showed that the cocci force themselves in between the epithelial cells as well as into the soft protoplasm, reaching as far as the substantia propria of the papillæ of the mucous membrane. After two days the whole epithelial layer may in this way be invaded by cocci. By that time there is also developed a very considerable tissue reaction, a number of white blood corpuscles having found their way out to the surface from the dilated capillaries through the epithelial layer. The epithelium of the limbus of the cornea, the border of the lid, and of the cornea itself, remains unattacked. After having caused greater or less destruction of the epithelium, the germs seem to lose their power of penetrating, and do not spread deeper than to the superficial layers of the sub-epithelial connective tissue, where their presence gives rise to the more intense inflammatory symptoms, and coincides with the purulent stage of the blenorrhœa. A regeneration of epithelium begins about the fourth day, and soon forms a protecting layer which, when eventually its outer portions lose their nuclei and have become converted into epidermis-like scales, offer an impenetrable barrier to the germs; and the discharge of pus, which continues during the whole time of the epithelial regeneration, comes to an end. That the gonococci are the essential cause of the inflammation was proved by the characteristic reaction which followed the introduction of a small quantity of a pure cultivation into the healthy urethra.

PHLYCTENULAR CONJUNCTIVITIS.—This is a distinct type of conjunctival inflammation, which, in its most characteristic forms, is almost entirely confined to children, in whom it is altogether much more common than in adults. The affection is a constitutional one, and an evidence either of struma or of some weakness following measles or other lowering illness of childhood,—often, indeed, of both combined.

Small clear blebs (phlyctenules), or in some cases pustules, surrounded by a pinkish injection, are met with on the conjunctiva covering the globe, or at the corneo-scleral margin. When in the latter situation, they are often found in considerable numbers if small, and may occupy different portions of the circumference at the same time. They may be so small as to be barely recognisable. When larger (2 mm. or more) they are either single or do not exceed two or three in number. The injection of the conjunctival vessels is usually limited to areas occupied by the phlyctenules, but is in some cases more general. Often there is at the same time phlyctenular keratitis, sometimes fascicular keratitis, and in long-continued severe cases, where there is a constant recurrence of the characteristic conjunctivitis, more or less marked pannus.

This disease is very apt to be associated with photophobia, a most troublesome complication, which, when not checked at an early stage, is apt to be long continued. It is a common thing to be told that an individual was "blind" for several months as a child. This blindness has in a large proportion of cases been merely that which results from the continual and convulsive closing of the eyes to which photophobia gives rise. Blepharitis, too, is a very frequent accompaniment, more common in connection with this than with any other form of conjunctivitis.

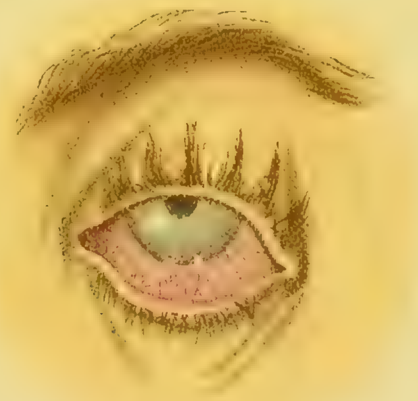


FIG. 23.—Phlyctenular conjunctivitis.

There is a marked tendency to relapses in phlyctenular conjunctivitis, many children suffering every year for many years in succession, and generally at much the same time of year, from attacks of the same nature. When this form of conjunctivitis occurs in adults it is only rarely that we find that it is for the first time. They have had "sore eyes" as children. Often the phlyctenules are absent altogether in the case of adults, and the affection is then only recognisable from the characteristic injection. Sometimes, indeed, in such cases there is a very striking resemblance between this affection and episcleritis, and some experience is required to make sure of the diagnosis.

The *treatment* of phlyctenular conjunctivitis should be both local and constitutional. In many cases the photophobia, too, calls for special treatment. Attention to the functions of the skin by daily bathing is very important; nourishing food, with plenty of milk, and cod liver oil whenever it can be tolerated, should be given. It is only exceptionally that cod liver oil, if properly administered in small quantities at first, or in the form of an emulsion, is altogether unsuitable. Locally the dusting into the eyes once daily of finely powered calomel, or the smearing into the conjunctival sac of a little Pagenstecher's ointment, and the occasional bathing with boracic acid lotion, is the treatment generally adopted. When there is much photophobia it is advisable to use atropine (0·5 per cent.) or cocaine (2 per cent.) as well, dropped into the conjunctival sac twice or thrice daily, after drying away the tears.

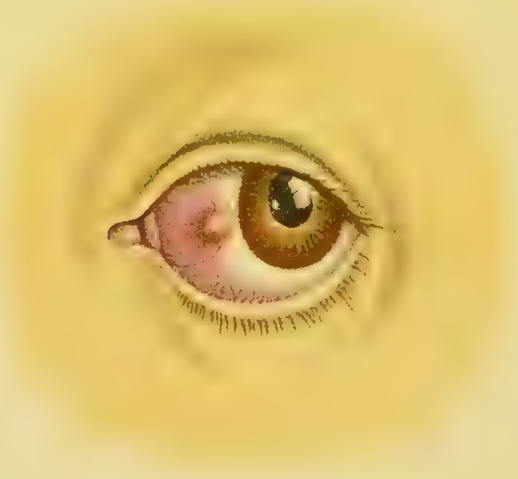


FIG. 24.—Pustular conjunctivitis.

Persistent photophobia in children is best treated by the old method of dipping the head into cold water and keeping it immersed for a few seconds, until the child begins to struggle for breath. The shock thus occasioned causes it to open its eyes, and there is afterwards not the same tendency to the spasmodic closing of the lids. Two or three days of this treatment is generally sufficient to check even a long-continued blepharospasm. Sometimes it is necessary to slit the outer canthus, or, where there is a marked contraction of the palpebral fissure, to perform the operation of canthoplasty, the effect of which is often extremely satisfactory. For the treatment of the accompanying blepharitis and eczema, see page 50. Adults, as a rule, do not stand the treatment with Pagenstecher's ointment as well as children. It is best in every case in the adult to begin trying it half strength (4 grs. to ℥i.). When calomel is used, the patient should not at the same time take iodine in any form internally, as a severe inflammation has been seen to be occasioned by the conversion of some of the calomel in the conjunctival sac into the very irritating biniodide of mercury.

Parents and others, as a rule, are in the habit of tying up, if they do not actually poultice, their children's eyes. To this practice is mainly to be ascribed the very considerable diffuse conjunctival hyperæmia and the eczema of the lids which one meets with in many cases seen for the first time. Tying up the eyes or keeping the patients in a dark room very much encourages photophobia, so that the practice is altogether to be discouraged. Plenty of light, open air, and exercise, are very essential elements in the treatment of phlyctenular conjunctivitis.

DIPHTHERITIC CONJUNCTIVITIS may exist alone, or along with diphtheria of the throat. In the latter case it would appear that the conjunctival inflammation is generally the first to make its appearance. This affection is very rare in Scotland though met with pretty frequently in some countries, especially North Germany.

The inflammation begins with hyperæmia and lachrymation, just as in simple catarrh, and rapidly proceeds to great swelling of the lids and chemosis, as in purulent conjunctivitis. The infiltration of the lids leads to greater stiffness and pain than in purulent conjunctivitis. A greyish-yellow membrane is not long in making its appearance on the palpebral conjunctiva. This membrane may cover only a portion of the surface of the conjunctiva, or may be found in patches over the surface. These patches sometimes become more confluent, and such cases are the most severe. The membrane cannot be completely removed without leaving a raw bleeding surface, which itself has not the natural healthy appearance, but presents that of a fibrinously infiltrated tissue. The discharge is less in quantity and much thinner than in purulent conjunctivitis, though it often afterwards becomes more copious and purulent.

There is danger for the cornea throughout, but especially during the first few days. The cornea is lost, as the result of a kind of necrotic and diphtheritic process combined, and is consequently often very rapidly destroyed. Ulcerations occurring at later stages are not so likely to lead to total destruction of the cornea. Constitutional symptoms, fever, and pronounced lassitude are met with in a much more marked degree than in other forms of conjunctivitis, but the affection rarely proves fatal unless there is at the same time a throat complication. Diphtheritic conjunctivitis appears to be most common in young children.

Various methods of *treatment* have been proposed. My own experience of this disease has been small. I have always adopted the treatment recommended by Tweedie of frequent bathings with a solution of quinine (3 grs. to ℥i.). This seems to be the treatment most generally followed in this country. Von Graefe recommended mercurial inunctions in the case of adults, and in Germany the local application and rubbing in of yellow oxide of mercury ointment is by some considered to have a decided effect in checking the progress of the inflammation. The question as to whether scarification of the conjunctiva, as practised often with advantage in purulent conjunctivitis, should be done in the diphtheritic form, is one on which there seems to be a want of unanimity. From the tendency to a specific infiltration of the wounds thus formed, it would certainly appear to be inadvisable. Constitutional treatment should, of course, be adopted from the first, and great care taken that portions of the membrane or of the discharge do not come in contact with other mucous surfaces, either in the patient or his attendants.

Granular Conjunctivitis, or Trachoma.—This form of inflammation is characterised by the appearance of granular-looking elevations in the conjunctiva, and by the subsequent cicatricial changes caused by the fibroid degeneration and loss of substance to which these give rise. It is met with in an acute as well as a chronic form, and at all ages, except in young children. Acute granulations may be the first beginning of the disease, or constitute merely an exacerbation of a previously existing and more or less quiescent chronic state of trachoma. The granulations, which are mainly, and in most cases indeed entirely, confined to the palpebral conjunctiva, are semi-transparent greyish or yellowish elevations. In every case they are met with of very varying sizes and arrangements, owing to their not all developing at the same time. Often they are more or less completely hidden by the swelling of the papillæ, to which they sooner or later give rise.

The actual nature of the granulations is a matter of dispute; most probably they are lymph follicles developed from the existing lymphatics, but they may possibly, as some suppose, be altogether new structures. It seems probable that they owe their existence to the presence of some specific microbe. This is, however, denied by many who are competent to form an opinion on the subject. According to Arlt, trachoma is altogether nothing but a form of chronic purulent conjunctivitis, owing its origin to the same infecting material as gonorrhœal conjunctivitis, but modified by having passed through a number of eyes in such a way as to be no longer capable of producing the same violent active inflammation. This view appears improbable on account of the absence of cicatricial formation, even in the chronic forms of purulent conjunctivitis, as well as from the existence in one disease of distinct granulations which are always absent in the other. At all events it is certainly of importance to make a distinct clinical difference between the two diseases.

When the disease occurs in its acute form, the very considerable lachrymation and photophobia, as well as the pain, cause attention to be at once directed to the lids, and the granulations are detected on everting them. An acute attack lasts in the most favourable cases for about a month. During the first week the granulations are most distinctly visible. They afterwards give rise to an acute catarrhal condition, by which they are not only at first masked, but apparently eventually absorbed or suppressed. Sometimes, however, the swelling of the conjunctiva subsides without having caused the disappearance of the granulations, and the disease thus passes into a more chronic form. In other cases it is chronic from the beginning, and the symptoms to which it gives rise may be so slight as to lead to little or no complaint on the part of the patient.

Chronic granulations last for months or years, and seem to have a tendency every now and then to set up an irritation which leads to acute catarrhal conjunctivitis. At other times one meets with cases, more especially such as have been properly attended to, in which the granular elevations are sparse and pale, but which all of a sudden are subject to an outbreak of fresh acute granulations. The extent, too, to which the papillary elements of the conjunctiva participate in the production of the abnormal appearance of the palpebral conjunctiva is very various. They may be uniformly enlarged to such an extent as to give rise to only a velvety-looking surface, more or less florid according to the degree of vascularity, or they may here and there assume the shape and appearance of warty excrescences. It is the condition of the conjunctiva, independently of the actual granulations, which determines the nature and amount of the secretion met with in any case of trachoma. The more swollen the conjunctival papillæ are, the more copious and muco-purulent is the secretion. In many cases there is indeed little or no secretion.

A first attack of acute granulations is only rarely accompanied by any corneal complication. Older cases, and especially such as are subject to frequent exacerbations, become, as a rule, sooner or later associated with pannus, which may involve the whole extent of the cornea, or only its upper part. The cicatricial contractions met with in all cases where the disease has existed for some time, present themselves in the shape of white bands, the most conspicuous of which run parallel with the border of the lid. There is, at the same time, more or less dryness and shrivelling up of the conjunctiva, which in extreme cases goes the length of complete destruction of the conjunctiva, a condition to which the name of *xerosis* is given. In long-continued cases of trachoma, too, the upper lids droop to some extent, giving a heavy appearance to the eyes, and one which, when it exists along with corneal opacities, is all but characteristic of the disease. The contraction which takes place in the conjunctiva leads in old cases to alteration in the direction of the eyelashes, distichiasis, trichiasis, and often, owing to curvature of the tarsus, to entropion.

The frequency of trachoma varies very much in different districts and communities. It is mostly met with in low marshy countries, and in places where the population is crowded, ill-fed, and under altogether unfavourable hygienic conditions. In foreign towns the Jewish, and in our own the Irish quarters, present the largest percentage of trachoma. In Scotland it is a rare affection except amongst the Irish population. Prisons and barracks, too, are, and more especially have been, visited by epidemics of it. Armies, particularly in the East, have been visited by the same disease, which, as general attention was first given to it on account of Larrey's description of the state of the eyes of the French army in Egypt in 1798, is often called *Egyptian Ophthalmia.* Egyptian ophthalmia includes, however, many other forms of conjunctival disease besides trachoma. There is no doubt that trachoma is contagious, though not highly so, and there seems good reason to suppose that the contagion may pass by the air in rooms which are ill-ventilated.

In the *treatment* of trachoma, attention must be given to cleanliness and ventilation. It is certainly inadvisable to allow any one with this disease to sleep in the same room with others, or to use towels or sponges in common with any one else. He should be well fed and kept as much as possible in the open air. In the acute form of the inflammation it is inadvisable, as a rule, to use astringents; at most, and only as long as the cornea is unaffected, a weak solution of acetate of lead (2 grs. to ℥i.), painted on the everted lids once daily, and boracic acid lotion as a wash may be employed. When on the supervention of catarrhal inflammation there is a good deal of muco-purulent discharge, nitrate of silver solution, preferably weak (5 grs. to ℥i.), may be cautiously applied to the everted lids. For the chronic cases there is certainly nothing better than bluestone. This should be applied twice weekly to the everted lids, taking care to get the crystal, which should be smooth, well at the retrotarsal fold. Some cases do not tolerate sulphate of copper; and, in such, touching with a smooth piece of alum, or painting with the solution of lead, may be used instead. I have no experience of the treatment with jequirity, which has lately been recommended. It is certainly unjustifiable to use it in cases where there is not very great defect of vision on account of pannus, as, so far, it has been found difficult, if not altogether impossible, to control the severity of the inflammation to which it gives rise.

Latterly attempts have been made to cure the more persistent cases of trachoma by excising altogether portions of the conjunctiva in which the granulations are most abundant. This treatment is much practised in Königsberg, where the disease is of frequent occurrence, and is strongly recommended by Jacobson. Sattler, too, who has had a large experience of the treatment by excising the conjunctival fold, speaks well of it, although he has given it up in favour of another method (*vide infra*). He considers that the chief objection which has been raised against the excision treatment, viz., that the cicatricial contractions thus caused might be worse than those resulting from the trachoma process itself, is not founded upon fact, but entertained only by those who have had no experience of it. An excision of a fold, 2 to 5 millimetres in breadth, produces absolutely no defect, and much more extensive excisions, undertaken in the more advanced cases of trachoma, do not cause the supposed interference with the normal movements, provided they be made superficially, and do not involve the tendon of the levator, or its insertion. When, however, in order to avoid making a too complete excision, and trusting to the favourable influence on the remaining follicles of a partial incision, groups of follicles are allowed to remain in the lower lid, conjunctiva bulbi, or caruncle, and the patient discharged before they have disappeared under suitable topical treatment, severe recurrences are far from being excluded.

In performing excision, however, not only is the specific new formation of the trachomatous process removed, but, in addition, other parts which, though perhaps they may not be in a normal state, are yet only secondarily affected in a manner from which they are quite capable of recovering. For this reason, and because the pathology of trachoma seems to afford a well-grounded indication for so doing, Sattler recommends the early and complete removal of the follicular deposits with as little interference as possible with the other tissues. The method employed by Sattler for this purpose consists in rupturing the follicles with a cataract needle, and scooping out their contents with a fine circular sharp spoon, 2 to 4 millimetres in diameter. In shelling out the contents of the follicles in the conjunctival folds, the tissues have to be put on the stretch by means of fixation forceps. All the follicles should be eradicated in this manner at one sitting, a general anæsthetic being used if necessary. The reaction is said to be usually inconsiderable, and the subsequent treatment consists in washing out the conjunctival sac with corrosive sublimate lotion.

The accompanying pannus does not call for special treatment, having a tendency to improve or disappear with the cure of the conjunctival inflammation. Other complications often require operative treatment, which is elsewhere discussed.

FOLLICULAR CONJUNCTIVITIS.—A not uncommon form of conjunctival inflammation, which presents very much the same appearance as is met with in some cases of granular conjunctivitis, and which by many is looked upon as a milder form, or even merely a different stage of that disease, is known as follicular conjunctivitis. The differential diagnosis between this and the granular form is by no means always easy, and often, at first, well-nigh impossible. The little elevations forming in the conjunctiva, the so-called follicles, have more the appearance of transparent blebs, whitish-yellow in colour. They are almost entirely confined to the lower lid, occurring, if at all, in much smaller numbers in the upper. They are associated, as in the case of the true granular inflammation, but never to the same extent as often happens in that disease, with swelling and hypertrophy of the conjunctival papillæ. The great clinical characteristic, however, is that they never lead to subsequent shrinking and cicatrisation; so that even should there be no anatomical distinction possible between follicular and granular conjunctivitis, the course of the affection sufficiently justifies a clinical distinction. Follicular conjunctivitis is mostly met with in children, and appears to be contagious. The affection lasts from a few weeks to many months. It appears to occur with equal frequency in districts which are free from trachoma as in those in which that affection is rife.

The *treatment* should consist in the improvement of the hygienic conditions; change of air, too, especially to some high-lying locality, where that is possible, is generally advisable. Where there is much catarrh, nitrate of silver and lead lotions may be used locally. When, on the other hand, the conjunctiva is abnormally pale and non-vascularised, the use of sulphate of copper (solid) is indicated.

AMYLOID DEGENERATION OF THE CONJUNCTIVA.—This is a rare disease, first correctly described by Von Oettingen of Dorpat, where it appears to be more frequent than elsewhere. Von Oettingen believed it to be an exceptional form of degeneration occurring in trachoma. Others consider it a separate disease altogether. In any case, it is clinically distinct, though the connection with trachoma is extremely probable. Thus, for instance, at Dorpat, where trachoma is common, amyloid degeneration is found to be most frequent between the ages of ten and thirty, which corresponds to the period of maximum frequency of trachoma. It is also met with in cases of old-standing trachoma. The appearance presented is that of yellowish and waxy-looking masses on the palpebral conjunctiva of both upper and lower lids, springing from the retrotarsal fold. The masses are usually very hard, and there is at the same time always a great enlargement of the tarsus. Portions excised give the characteristic reaction of amyloid degeneration. The exact nature of the process is differently described by various histologists. There seems little doubt, however, that the process is entirely a local one, as it is rarely, if ever, associated with similar degeneration elsewhere. The *treatment* recommended is the excision of the waxy masses along with the tarsus.

Essential Shrinking of the Conjunctiva.—Occasionally, and without any marked inflammatory symptoms at the time, an atrophy of the conjunctiva, or a complete growing together of its opposed surfaces, takes place. The connection between this degenerative change, to which Alfred Graefe has given the name of essential shrinking of the conjunctiva, and pemphigus, was pointed out a few years ago by Steffan. Cases of pemphigus of the conjunctiva had previously been described, in which the eruption on the conjunctiva formed part merely of a more or less generally distributed skin affection of the same nature. In such cases, too, it was observed to lead to shrinking. In the cases of essential shrinking it has been supposed that the pemphigus may be limited to the conjunctiva. Cohn, for instance, has published a case where the suspected connection between the shrinking of the conjunctiva and pemphigus was afterwards confirmed by the development of a characteristic eruption all over the body. In this country, cases of this peculiar conjunctival disease have been described by Lang, Critchett, and Nettleship, which, as well as others reported from the clinics of Schweigger and Alfred Graefe, render the connection between the two conditions extremely probable. Several cases have come under my own observation, but I have only once seen the disease accompanied by pemphigus. In that case, too, the skin affection was not a true pemphigus, but apparently more an eczema with occasional large blebs, decidedly a mixed form.

The *treatment* of essential conjunctival shrinking has been always without any effect, even where transplantation of mucous membrane from other parts has been tried.

SUBCONJUNCTIVAL ŒDEMA.—More or less pronounced swelling of the conjunctiva, without any local inflammatory redness, giving the appearance of a semi-transparent bleb or bulla surrounding the cornea, and it may be protruding from between the lids, is met with in cases of inflammation in the orbit, eyelid, or nose. When there is no focus of inflammation in the proximity, it is usually a symptom of some general affection—heart, kidney, &c.—and occurs along with œdema in other parts of the body.

Occasionally one meets with œdema of the conjunctiva evidently unassociated with any other affection. Thus, I have seen several cases in which a non-inflammatory chemosis recurred at intervals, or while permanently present to some extent increased in severity at different times, in otherwise perfectly healthy individuals. In one case recurrence of excessive œdema of this nature had taken place from four to six times every year for eighteen years, the first attack being rightly or wrongly ascribed to a snake-bite in India.

ECCHYMOSIS.—Any rupture or incision of the conjunctiva, produced either accidentally or by some operation, gives rise to effusion of blood into the membrane, which, according to its extent, takes from one to three weeks to undergo absorption. Spontaneous ecchymoses occur by the bursting of conjunctival vessels, usually owing to some excessive strain which has caused temporary congestion of the head—coughing, sneezing, vomiting, &c. A tendency to the occurrence of such ecchymoses indicates often a degenerated state of the vessels, and may therefore be of symptomatic importance.

EMPHYSEMA of the conjunctiva may occur alone or along with ecchymosis. In the latter case, some connection has been established between the air passages and the orbital tissues. The condition is rare.

PTERYGIUM.—An inflammatory or hypertrophic thickening of a portion of the conjunctiva, of a triangular shape, firmly attached by its apex of the superficial layers to the cornea, has received the name of pterygium, owing to its fancied resemblance to the wing of an insect. The sides of a pterygium form distinct folds, under which a probe may be passed, often for a considerable distance. The folding-in of the sides gives an appearance as if some new membrane lay over the conjunctiva. Very considerable differences exist in the consistency of the pterygium. In some cases it is thin, pale, and fibrous; whilst the other extreme is represented by a swollen, red, fleshy elevation of the characteristic triangular form.

The ordinary pterygium is always found in a position corresponding to the palpebral fissure, and therefore over one of the lateral recti muscles; much most commonly over the internus. Sometimes it exists in both eyes, and occasionally two are met with in the same eye.

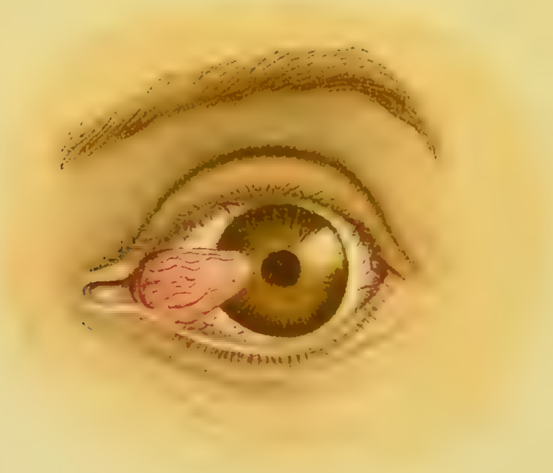


Fig. 25.—Pterygium.

Pterygium has a tendency in most cases to spread over the cornea, though it is rarely seen to extend beyond the centre of the pupil. It occurs only in individuals who are exposed to constant irritation of the conjunctiva, and in whom, at the same time, the conjunctiva is tolerably lax. It is therefore most frequently met with in masons, field labourers, colliers, &c., and generally after the age of forty. The irritation to which they are subjected, causes, in all probability, not only hypertrophy of those portions of the ocular conjunctiva, which is not covered by the lids, but a loss of the normal epithelial covering, so that there is a tendency for an adhesion to take place between the conjunctiva and the epithelial surface of the contiguous cornea, the vitality of which is also interfered with from the same cause. After such an adhesion has once taken place, the continuance of the irritation causes the surface of the conjunctiva to double over more and more on the cornea, and thus to extend its attachment with the superficial layers of the cornea, and at the same time drag more and more on the surrounding conjunctiva. A less common form, or what may be called a *spurious pterygium*, is met with at any part of the conjunctiva. It is the result of the attachment of a portion of chemotic conjunctiva to a subjacent ulcer of the cornea. Such cases are only met with after severe inflammations, and do not extend. They also, as a rule, leave a complete space, which is bridged over, so that a probe may be passed from side to side below the fold of conjunctiva.

There does not appear to be any connection between the action of the ocular muscles and the position usually assumed by a pterygium, that being probably entirely dependent on the conjunctiva opposite the palpebral fissure being most subject to the changes which long-continued irritation produces.

The *treatment* to be adopted in the case of pterygium should depend upon whether or not it is progressing in such a manner as to interfere with vision or with the free mobility of the eye. If this be the case, or if for the sake of appearance the patient wishes it to be removed, an operation should be performed. The most suitable and satisfactory method of operating is that described in Chapter XVIII. When this is properly done it is rare to meet with any recurrence. It is essential, however, in order to insure success, that the patient should not be exposed to the same irritation to which the original occurrence of the affection is to be ascribed, until some time after complete healing has taken place. When possible, it is advisable that he should change his occupation altogether for one which does not entail the same risk. In cases where, although there can be no doubt as to the gradual progression of the pterygium, the patient is nevertheless unwilling to submit to operative interference, he must be protected against the constant irritation in some manner best suited to the requirements of each case. If he continues his wonted occupation the eye should be tied up for a lengthened period. The spurious form shows little or no tendency to progress, and never recurs after operation.

PINGUECULA.—Another change met with in the ocular conjunctiva of adults, as the result of continued irritation from some cause or other, is what is called pinguecula. This is a whitish or pale yellow condensation of the conjunctival tissues covering the sclera mostly to the inner and outer sides of the cornea, and corresponding, therefore, to that portion which lies opposite the palpebral fissure. The patch is often irregularly triangular in shape, and generally only very slightly prominent. Most commonly it is met with to the inner and outer side in the same or in both eyes, but more pronounced, as a rule, to the inner side. It does not cause any inconvenience, and does not call for operation. Pinguecula is a slow inflammatory thickening of the deeper tissues of the conjunctiva, which, although yellow in appearance, does not seem to entangle any fat.

INJURIES TO THE CONJUNCTIVA — FOREIGN BODIES IN THE CONJUNCTIVA.—Clean cuts in the conjunctiva readily heal, if the edges of the wound be brought together by stitches, and without giving rise to inflammatory reaction. Left to themselves, such wounds are apt to gape, so that healing takes place by granulation, and is therefore often accompanied by more or less conjunctivitis. Foreign bodies when retained in the conjunctival sac for days or weeks set up a traumatic conjunctivitis. They usually lodge somewhere under the upper lid, and mostly become fixed, with or without penetration, in some portion of the conjunctiva covering the tarsus, often close to the lid margin. Sometimes they become embedded in the folds of the retrotarsal portion of the conjunctiva, or owing to suction—as, for instance, in the case of husks of corn—lie with the concave surface against the conjunctiva. In the first position they are apt to give rise to ulceration of the cornea. Owing to this circumstance, it is well to make a practice of always everting the lids, and examining the conjunctival surfaces carefully in all cases of inflammation of conjunctiva, or abrasion or ulceration of the cornea. Portions of glass are the most difficult to detect, so that when there is any suspicion of there being a foreign body of this nature present, the everted lid should be examined with oblique illumination, and, if necessary, with a magnifying glass. Besides the cutting and contusion which may be caused by mechanical injury to the conjunctiva, and the subsequent changes, inflammation and cicatrisation, which result in this way, other and often more serious changes are caused by agents which have a chemical or thermal action.

Accidents from the introduction into the eye of acids, alkalies, lime, boiling water, or molten metal are not uncommon. As a rule such accidents are immediately followed by great pain, which leads the patient to seek advice as soon as possible. When, as usually happens, a portion of the conjunctiva has been destroyed, the prognosis will depend greatly on the extent as well as position of the injured portion. Except where the cornea is seriously damaged at the same time, the worst cases are those in which a considerable extent of both ocular and palpebral conjunctiva has been destroyed, and more especially when the destruction has involved the retrotarsal fold. It becomes, then, impossible to prevent adhesion of the lid to the globe, or what is called symblepharon. A symblepharon necessarily, too, gives rise to more or less restriction of the movements of the eye. It is often possible to remedy the defect by some operation; but it is of great importance to make an effort in all cases seen soon after the accident to avoid the formation of an adhesion between the two raw surfaces. This can, as a rule, be successfully accomplished, if the fold of transition from the ocular to the palpebral conjunctiva be not injured to any great extent, by frequently dropping a few drops of olive oil into the eye after washing it out with a weak non-irritating antiseptic lotion (corrosive sublimate, 1 in 5000, or a saturated solution of boracic acid), and at the same time frequently separating the two surfaces. This treatment should, in any case, be adopted immediately after an injury has been received which is likely to cause destruction of the conjunctiva; but it must, of course, always be preceded by a thorough examination of the whole extent of the conjunctival surface, and the removal of any foreign bodies. In cases where the burning is the result of acid, it is advisable, in order to neutralise any of the acid which may still be present, to bathe with a weak solution of washing soda, which is generally at hand.

Electric Light Ophthalmia.—A new form of conjunctivitis is now occasionally met with since the strong arc electric light has come so much into use. Amongst those whose eyes are subjected to the glare of a powerful arc light, an intense conjunctival irritation is sometimes found to come on several hours afterwards. The condition is exceedingly painful, accompanied by swelling of the lids and great photophobia. It has been shown by Widmark, by a series of beautiful experiments, that it is the violet and ultra-violet rays contained in the electric light which cause the characteristic irritation.

Snow-blindness. — Both as regards its symptoms and etiology, snow-blindness is closely allied to the form of conjunctival irritation set up by exposure to strong electric light. There is intense photophobia, swelling of the lids and chemosis, following upon the exposure for some hours to the light reflected from snow. As in the case of electric light ophthalmia, it is the irritation of the conjunctiva by the shorter, more actinic waves of light which gives rise to these symptoms. According to some, they are caused as well by exposure to sharp, frosty air containing minute ice spicules.

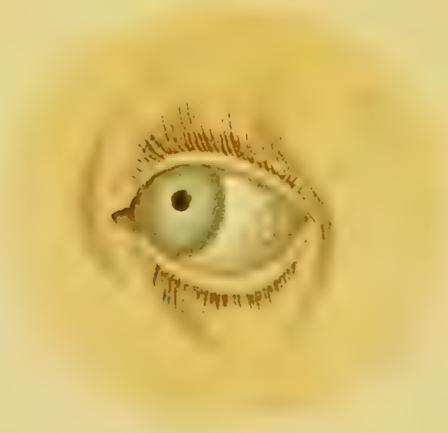


Fig. 26.—Lymphangiectasis of the conjunctiva.

Dark smoked glasses should be used as a protection wherever there is the risk of either electric light or snow-blindness.

Lymphangiectasis of the Conjunctiva.—A not uncommon appearance is met with in the conjunctiva, as the result probably of some interference with the normal flow of the lymph, and consequent dilation of the lymph spaces. This change, which seems to be of no importance, and not associated with any inflammation, consists in the formation of blebs of clear straw-coloured fluid, not bigger than a pin's-head, but crowded together in masses. The blebs are situated in the superficial layers of the conjunctiva, so that they can be readily pushed with the conjunctiva over the subjacent tissues (see Fig. 26). They often disappear spontaneously after some weeks or months.

TUMOURS AND OTHER AFFECTIONS OF THE CONJUNCTIVA.—Tumours of the conjunctiva are rare. Of the non-malignant forms perhaps the most common are papillomata. They usually occur as multiple excrescences from the conjunctiva, at the inner angle of the eye in the region of the caruncle, but are found at the same time springing from the palpebral conjunctiva. Their surfaces are generally uneven, often crenated, but they may also be smooth. If thoroughly removed there seems to be no tendency to recurrence.

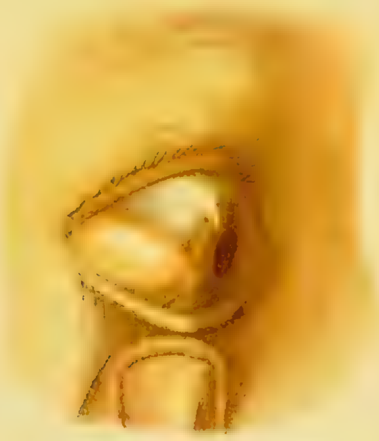


FIG. 27.—Dermoid of cornea.

Simple enlargement of the caruncle is sometimes met with.

Dermoid Cysts of the conjunctiva are situated at the corneo-scleral margin, and involve the cornea as well. (Fig. 27.)

Simple Cysts, congenital and traumatic, are described as having been met with; also others containing a cysticercus which sometimes makes its appearance subconjunctivally.

Of malignant tumours *sarcomata* of the conjunctiva are not so very uncommon. They are most frequently met with in the ocular conjunctiva. They may or may not be melanotic. Even when apparently very completely removed, they are extremely liable to recur. When, therefore, no doubt exists as to their nature, as, for instance, when they are melanotic, and when in catching hold of them with the fixation forceps they readily break and bleed, and show themselves to be of a soft buttery consistence, the best treatment, unless the other eye should be blind or absent, is to remove the eye, and at the same time a part or all of the contents of the orbit.

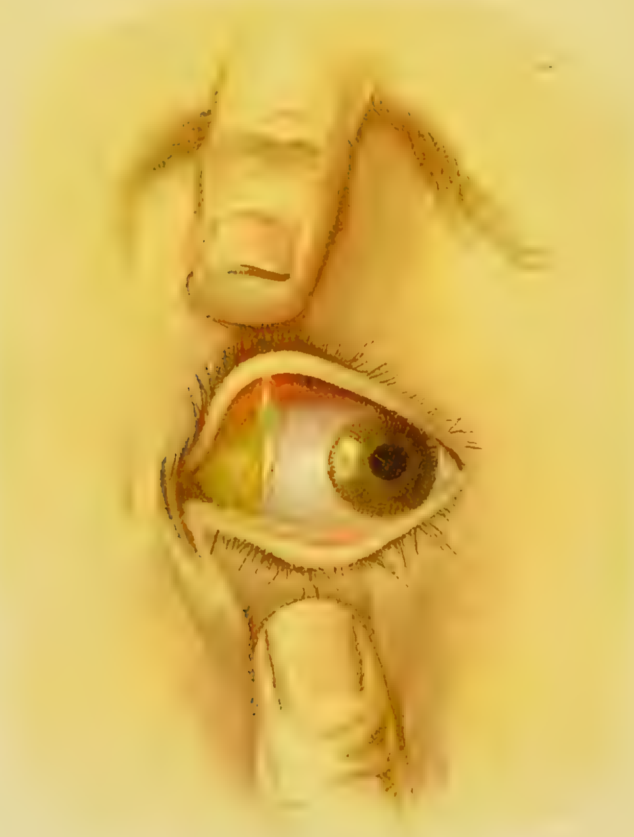


Fig. 28.—Chancre of conjunctiva.

Rodent ulcers and *epitheliomata* are generally found as extensions from the skin of the lids, but they may originate in the conjunctiva. They must in any case be freely removed.

Tubercular and *lupoid swellings* of the conjunctiva occur as rare affections, the latter mostly spreading from the skin, while the former appear to originate in the conjunctiva. They both have a tendency to spread to the cornea. The differential diagnosis is by no means easy. The so-called tubercle bacilli have been found in some cases. Cases which I have examined for this microbe have all led to negative results, although they have sometimes proceeded to complete destruction of the eye. Some such cases, as far as my own experience goes, appear to be more of the nature of strumous thickenings and ulcerations than real tubercular masses. The *treatment* consists in excising or thoroughly scraping away the soft tumour tissue.

CHANCRES occur occasionally in the conjunctiva, and involve the skin of the surrounding lids. They are not, as a rule, difficult to diagnose, but any doubt is set at rest by the appearance of secondary symptoms in a certain proportion of all cases.

HYPERPLASTIC SUBCONJUNCTIVITIS.—I have met with several cases of a condition which I have not found described elsewhere, and which appears to be a hyperplasia of the deeper tissues of the conjunctival fold. It seems always to occur in the lower lid, and forms a hard mass of almost cartilaginous consistency, which springs immediately into view when the lid is everted. It may even grow so large as to project beyond the lid. It is associated with but little conjunctival irritation, though sometimes producing œdema, and disappears after some months without treatment. Fig. 29 is from a case where the condition existed in an aggravated form.

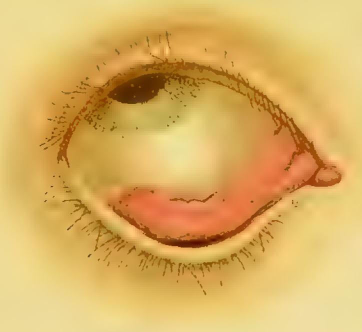


FIG. 29.—Hyperplastic Subconjunctivitis.

EPISCLERITIS.—The deeper tissues covering the sclera are subject to a characteristic type of inflammation, to which the name of episcleritis is generally given. Possibly sometimes the superficial layers of the sclera itself may also be involved, but in most cases the inflammation is mainly or entirely confined to the looser tissues above it.

Episcleritis shows itself as a patch of inflammation, the most intense point of which is generally situated some millimetres from the corneo-scleral margin. In appearance the patch is at first not unlike that caused by the injection surrounding a large conjunctival phlyctenule. Its colour is, however, darker, being of a deeper red, with not infrequently a tinge of violet. The patch is always more or less prominent, sometimes very markedly so, and gives rise then to the appearance of a considerable bulging of the sclera in the area of inflammation. In size it varies much in different cases; but, while generally considerably less, may occasionally involve one-fourth of the extent of the ocular conjunctiva. After the inflammation has subsided, it leaves more or less trace of its former existence in the shape of a steel or slate-grey patch, which generally merges into the surrounding healthy coloured conjunctiva.

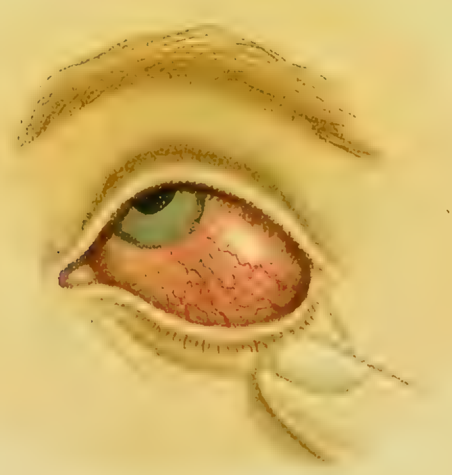


FIG. 30.—Episcleritis.

As a rule, episcleritis does not cause too much spontaneous pain, though there is always more or less tenderness to touch over the inflamed patch. Sometimes a dull heavy pain is complained of, and there may even be a considerable degree of photophobia and lachrymation. It is a common enough affection, confined almost entirely to adults, and occurring with equal frequency in both sexes.

The inflammation runs a very chronic course, several months usually elapsing before the inflamed patch altogether disappears. It is subject both to exacerbation from time to time during its existence, and to recurrence. When recurrence takes place, another similar patch forms at some other part of the episcleral tissue, and not infrequently a second patch may make its appearance before the first has entirely disappeared. The most common position for the first patch seems to be to the outer side of the cornea, but it may be anywhere. When very near the cornea some infiltration may pass into that membrane and consequently produce an opacity, but this almost always clears away afterwards. As a rule, the cornea is altogether unaffected.

Episcleritis is a decidedly gouty or rheumatic affection, and, so far as my experience goes, such a diathesis is the only one with which it can be connected. Some writers consider it, but without, I think, sufficient reason, a manifestation of syphilis.

In the *treatment* it is better to avoid any local application; certainly all astringents are more likely to do harm than good. Gentle massage, performed by rubbing the lid rapidly over the inflamed area for half a minute to a minute at a time, once or twice a day, or oftener when it is not very sensitive is useful, and seems to promote absorption of the infiltration. Salicylate of soda may be given internally, In the more distinctly gouty cases colchicum is often better. When there is much tendency to recurrence the patient should be sent to some bath, such as Harrogate, Buxton, or Aix-les-Bains. It is seldom that any surgical interference is called for, and nothing should be tried except in cases which are very chronic, *e.g.* when the first patch of inflammation remains for six months or more without any improvement, and becomes frequently reinflamed and associated with corneal infiltration. In such cases I have had excellent results from removing a portion of the inflamed tissue right up to the corneo-scleral margin, that is, by performing the operation of peritomy or syndectomy. Incision of the swollen area, when it is very prominent, and scraping out of its contents, has been recommended, but of this treatment I have had no experience.

CHAPTER IV.

DISEASES OF THE CORNEA.

General Remarks on Inflammation of the Cornea.

The corneal tissue may be the site of inflammatory products, deriving their origin from a focus of inflammation which is situated either in the cornea itself, or in some other part of the eye. In the first case we may talk of a *primary* or true *keratitis;* in the second there is, properly speaking, no keratitis, but merely a more or less dense, diffuse infiltration of the cornea, similar in every way to the hyperæmic and œdematous area of infiltration which surrounds a focus of inflammation in any other part of the body. The condition is one, then, of secondary diffuse infiltration of the cornea; secondary as distinguished from a similar infiltration surrounding a primary inflammation of the cornea; or, shortly, *secondary keratitis,* if it be remembered that the secondary refers to the focus of inflammation being elsewhere, and not to the time at which the corneal changes become manifested.

The division of corneal inflammatory changes into primary and secondary keratitis is the most important classification from a clinical point of view.

Primary keratitis, except when confined to the epithelial layer, is mostly associated with some degree of destruction of the corneal tissue at the focus of inflammation, and the loss thus sustained is replaced by more or less intransparent connective tissue. It occurs in two forms—as an *infiltration* or *abscess,* and as an *ulceration;* that is to say, the inflammatory changes are either surrounded by more or less healthy tissue, or have led to a destruction of the superficial layers of the cornea, so that there is an open sore.

It is customary to distinguish between primary infiltration and abscess of the cornea. The latter is said to exist when the infiltration is purulent and destruction of the tissues has taken place. It is not always possible to make this distinction clinically. The colour of a corneal infiltration depends so much on the depth at which it lies as well as on its density, that if one were to judge by colour alone, one would frequently diagnose an abscess when such did not exist in the anatomical sense. The degree of pain is also a very uncertain symptom, as great differences exist in this respect.

Clinically, then, we may divide primary corneal inflammation into *ulcerative* and *non-ulcerative.* The non-ulcerative frequently becomes eventually ulcerative. Both forms may be *single* or *multiple,* according as one or more foci of inflammation are present. They may be *superficial* or *deep, diffuse* or *circumscribed, vascularised* or *non-vascularised,* and may or may not be complicated by inflammation or other changes of any other part of the eye, such as iritis, scleritis, or any alterations in the contents of the aqueous chamber, such as the presence of pus (hypopyon), or blood (hyphæma).

An ulcer of the cornea may extend either in depth or in breadth, or in both. When extending in breadth, the margin, or some portion of the margin, is ragged and usually visibly infiltrated, and the base covered with more or less débris. Sometimes, and this is more especially the case with ulcers near the margin of the cornea, a number of contiguous foci of inflammation become confluent, and give rise to one large ulceration.

When healing takes place, the margin of an ulcer becomes rounded all over, owing to the extension over it of an epithelial covering, and the loss of substance is gradually repaired by the development of an intransparent connective tissue. There is thus left an opalescent or white opacity, a so-called *macula* or *nebula* of the cornea, as the result of the cicatrisation. An exceedingly dense white cicatrix is called a *leucoma.*

A nebula of the cornea does not clear up in the adult, but does so in young children, and the more readily, the younger the subject. The cicatricial tissue of which they are constituted then gradually assumes a greater and greater similarity to the healthy transparent tissue. Under the most favourable circumstances, viz., when the corneal destruction takes place in infancy, and the child is otherwise healthy, all but the most extensive and deepest ulcerations may in this way eventually leave no trace of their existence.

An ulcer which extends in depth may lead to perforation of the cornea. This may give rise to further complications, the nature of which depends on the situation and extent of the perforation. If small and central, there may be no further complications than the emptying of the anterior chamber, whereby the lens comes in contact with the posterior surface of the cornea. When the lens remains long in this position there is apt to be set up a proliferation of the cells lining its anterior capsule, leading to what is called *anterior capsular* or *pyramidal cataract.* When the central perforation is large, the lens, and even more or less of the vitreous humor, may be expelled through it. Sometimes a perforation at the centre of the cornea remains for a long time open, constituting what is called a fistula of the cornea. When a perforation occurs more towards the margin of the cornea, it is the iris which, on the escape of the aqueous humor, becomes applied to the posterior surface of the cornea. The most favourable result which may follow this accident is for the iris to go back into its proper position on the re-establishment of the anterior chamber. This happens if the perforation is small and rapidly closed. The portion of the anterior surface of the iris, which at the time of perforation becomes pressed up against the back of the

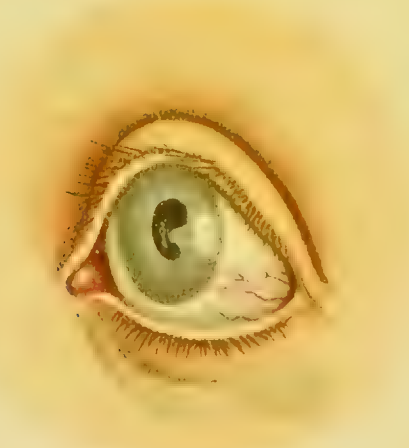


FIG. 31.—Anterior synechia.

cornea, may form a permanent fibrinous adhesion in this situation, and thus give rise to what is called an *anterior synechia* (see Fig. 31). There is then more or less interference with the free movement of the pupil, which, as it dilates less in one or more directions than in others, loses its circular form and becomes irregular in shape, usually oval or egg-shaped. In the case of larger perforations, the iris is entangled in the corneal opening, and more or less of it may protrude or prolapse externally. The iris usually then becomes incorporated in the cicatrix. To this condition the name of *leucoma adherens* is given.

Corneal ulceration, when extensive or deep, leads also to alteration in the curvature of the cornea. Such an alteration may or may not be accompanied by more or less marked protrusion of the cicatrised portion, or what is called *staphyloma* of the cornea. A corneal staphyloma may be partial or complete, according as only a portion or the whole of the cornea is involved in the protrusion.—(See Figs. 32 and 33.) The most severe cases of corneal destruction are followed by disappearance of the cornea—*phthisis corneæ.* Not infrequently a corneal inflammation is associated with, or leads to, inflammation of the iris.

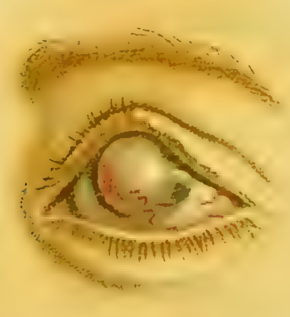


Fig. 32.—Large staphyloma of cornea.

The principal *causes of primary keratitis* are traumata, either with or without subsequent sepsis, struma, and extension of conjunctival inflammations. The homogeneous elastic layer of Descemet, which, along with its layer of epithelium, bounds the true corneal tissue posteriorly, does not undergo inflammatory change. When perforation takes place after there has been an ulcerative destruction extending through the whole depth of the superficial layers, it is owing to the bursting of this membrane, which may for some time previously have been protruded into the ulcer, giving rise to the condition known as *keratocele.* Any sudden strain, such as may be caused by coughing or sneezing, leads to the rupture, which often occurs with some violence, the aqueous humor being squirted out through the opening. The sudden relief of tension caused by the evacuation of the anterior chamber is not followed by any serious consequences if the deeper tissues of the eye be healthy. After perforation there is usually observed a great tendency to healing, a fact which it is sometimes advisable to take advantage of by performing paracentesis of the cornea through the base of the ulcer.

The *treatment* applicable for most cases of keratitis consists in keeping the cornea as free as possible from external sources of irritation. Strong light should be avoided by the use of a shade covering both eyes, or a pair of smoked-glass spectacles. The patient should not be allowed to read or use the eyes much for any work near at hand. He should be protected from dust, smoke, or close atmosphere of any kind. Weak antiseptic lotions, preferably of corrosive sublimate (1 in 5000) or boracic acid (1 in 50) may be used frequently to bathe the eyes with. Poulticing should, as a rule, be avoided, although in some cases warm antiseptic fomentations are comforting. When there is much injection and photophobia the pupil should be kept dilated with atropine, in order to avert any complication with iritis, or to render the consequences of such a complication less serious than they might otherwise be. Some forms of ulceration, more particularly the sluggish non-vascularised forms, appear to heal more rapidly when eserine drops (2 grs. of the sulphate to ℥i.) are used three or four times daily. Strumous affections of the cornea are much benefited by stimulation with yellow oxide of mercury ointment.

As a general rule, and unless the ulcerations be very deep or very extensive, so that there is danger of perforation, or unless the movements of the lid evidently produce irritation, it is better not to tie up the eye, as more harm is done by the retention of the secretions and tears than by free exposure to the air.

When the scar which remains after healing is dense and situated in front of the pupil, vision may sometimes be greatly improved by the performance of an iridectomy. The most favourable situation for an artificial pupil is inwards, or inwards and downwards, but it should be made opposite that portion of the cornea which is most transparent, and at the same time least altered in its curvature. Before performing the operation, the cornea should therefore be carefully examined by oblique illumination, and with Placido's or other similar keratoscope. Owing to the extent to which some opacities clear up, it is not advisable to perform iridectomy, for merely optical reasons, too soon. A little practice enables us to determine what part of the nebula remaining is likely to clear. That portion which is densely white and sharply defined will, except in the case of very young children, always remain; but the more diffuse area, which at first generally surrounds this, and which does not correspond to the portion in which there has been any actual destruction of tissue, is likely to clear away. When this lies over the pupil then, it often happens that the eventual acuity of vision is found to be rather less than it would otherwise have been if an iridectomy had not been performed. In any case, even when one has to do with a permanent opacity, it may be taken as a good practical rule that iridectomy should not be performed unless the vision is less than $\frac{20}{200}$. An iridectomy, performed for optical purposes, should be small, and the incision should be made as a rule at the corneo-scleral margin.

Something may often be done to promote the disappearance of nebulæ, more especially when they are the result of tolerably superficial inflammations. Either massage alone — that is, rapidly rubbing the lid over the eye for half a minute at a time, two or three times daily, or that, combined with yellow oxide of mercury (Pagenstecher's) ointment, is useful in this respect. Another plan is to drop into the eye daily, or once every second day, a drop or two of turpentine, or turpentine combined with an equal amount or more of olive oil. Such stimulating treatment should not be begun too soon, and should be stopped when it produces much irritation, as there is some tendency for fresh infiltrations to take place in, or in the immediate neighbourhood of, old cicatrices.

When there is a staphylomatous protrusion of the scar, a good-sized iridectomy should be performed, and in such a situation as to free, if necessary, any dragging on the iris (see Fig. 33). The result of iridectomy performed for this purpose is often most satisfactory. The dragging on the iris leads apparently to increased secretion of the fluids within the eye, and consequently to a rise in the tension of the eye, which the more or less weak cicatricial tissue is unable to withstand. Iridectomy used also to

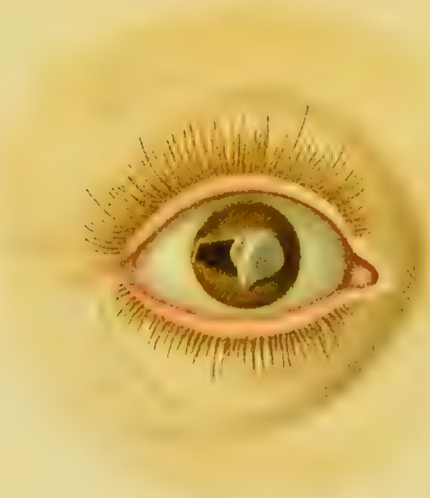


Fig. 33.—Case of partial central staphyloma of cornea, for which iridectomy has been performed.

be performed for cases of anterior synechia which were unaccompanied by any staphylomatous protrusion of the corneal cicatrix. Its was supposed that the anterior synechia entailed a risk of sympathetic inflammation from irritation, which might at any time be set up, owing to dragging on the iris. As a matter of fact, however, the risks of sympathetic inflammation are increased instead of diminished by iridectomy. Such an accident is not only extremely rare in the case of a simple anterior synechia, but only occurs at all if, for some cause or other, a septic infiltration of the scar takes place. No doubt the dragging on the iris sometimes does set up irritation, but even this is, comparatively speaking, an exceptional result. The proper treatment is to leave the eye alone, and only interfere if there be irritation. A double iridectomy, freeing the iris on either side of the adherent portion, should then be performed. Both may be done at the same time without difficulty. Attempts at detaching the adhesion by any operation should not be made. They are rarely, if ever, satisfactory.

Larger staphylomata may be reduced in size by any of the operations described in the chapter on Operations. There is some risk of setting up a prolonged irritation or inflammation by such operations, which at the same time introduce the possibility of sympathetic inflammation in the other eye. Unless very unsightly, a total or nearly total staphyloma is therefore best left alone. When protruding between the lids and painful, evisceration or enucleation should be performed in preference to other less severe measures, but as long as the tension remains high there is hardly any, if any, danger of sympathetic inflammation.

Leucomata may be rendered less unsightly by being tattooed Sometimes, too, when the cicatrix in the cornea covering the normal pupil is not very dense, the effect of an iridectomy is increased by this operation. The vision is thereby increased in acuity, being hampered by light scattered by the corneal opacity. Careful tattooing, without iridectomy, sometimes leads to very considerable improvement of vision in cases where the whole pupil is not covered by cicatrised cornea.

SPECIAL FORMS OF PRIMARY KERATITIS.

PHLYCTENULAR KERATITIS.—This very common affection is not properly characterised by the name which it most frequently gets. But as it is the process in the cornea which corresponds to phlyctenular conjunctivitis, with which it is very frequently associated, there seems no reason to prefer such names as scrofulous or lymphatic keratitis, or herpes, or eczema of the cornea, which have been by some substituted for it.

Phlyctenular keratitis occurs in the form of single or multiple, small, superficial, and, at first, usually non-vascularised, infiltrations of the cornea. The size of these infiltrations is rarely more than a pin's-head. They are of a greyish colour, and slightly prominent. Frequently they break down into small ulcers, which do not as a rule exhibit any tendency to spread either superficially or in depth, but which frequently become vascularised. A slight nebulous spot remains for some time after healing has taken place, but eventually disappears entirely. Sometimes the little infiltrations are decidedly yellow or purulent, and break down into ulcers, which lead to destruction of some of the true corneal tissue, and therefore to more permanent nebulæ. A certain, and often considerable, amount of pinkish circumcorneal injection is met with in this affection, and there is at the same time almost invariably a good deal of lachrymation and photophobia. The photophobia is often so intense in children as to lead to most persistent blepharospasm. The most common cause of phlyctenular keratitis is struma, but it is often met with where there exists some constitutional depression not necessarily of a strumous nature, such as weakness after exhausting illnesses, measles, scarlet fever, &c. In strumous children it is very apt to recur once a year or oftener for some time. Altogether the affection is much more common in children than in adults, and usually, when met with in adults, there is a history of previous attacks in childhood.

Except in more markedly purulent cases, the *treatment*, in children at least, should consist mainly in the local application once daily of the yellow oxide of mercury ointment (Pagenstecher's) of the strength of 8 grs. of the yellow oxide to ℥i. of unguentum cetacei, to which a few drops of olive oil may be added to keep it from becoming hard. The spermaceti is better than vaseline, which sometimes irritates. This ointment is certainly better than any other form of mercurial preparation. Whether this is owing to its stimulant or its antiseptic properties, or to some other cause, is not very evident. Adults do not tolerate it so well as children. They require altogether less stimulating local treatment; atropine, cocaine, and weak antiseptic applications, such as a 2 per cent. boracic lotion, or a lotion of corrosive sublimate, 1 in 5,000 or 10,000. The internal administration of cod liver oil is of great service in children. Attention should also be given to keeping the skin clean. For adults, quinine, or, in the strumous, the syrup of the iodide of iron, are indicated. Tying up the eye is not only unnecessary, but does harm, partly on account of encouraging the photophobia, and partly owing to the poulticing action which is apt to be caused by the bandage or cloth when saturated with the tears which are usually copious in this affection. At most a shade may be allowed on account of the photophobia, but it is better as a rule to try in every way to combat this condition.

The photophobia is sometimes so severe, and at the same time so out of keeping apparently with the actual severity of the inflammation, that it must be looked upon more as an induced neurosis which calls for special treatment. Cocaine and atropine are useful as anodynes, and in cases where the blepharospasm is very marked and prolonged, division of the outer canthus may be practised. In children the plunging of the head into cold water has often a marvellous effect, especially if it be held sufficiently long in the water to give rise to a gasping for breath. Counter-irritation, either by blisters or setons in the temples, or by the application of nitrate of silver to the skin of the upper lids, is unnecessarily severe, and at the same time not often a very successful treatment, though still apparently pretty much used.

FASCICULAR KERATITIS.—Closely allied to phlyctenular keratitis etiologically, and indeed often occurring along with it, but constituting a distinct clinical type of corneal inflammation, is what is called fascicular keratitis. This is a vascularised infiltration, the vessels in which run parallel to each other, so as to form a dense band or fasciculus about a line in breadth stretching into the cornea (see Fig. 34). At the end of the vascular leash the infiltration is somewhat more or less crescentic in shape, and often ulcerated. Occasionally two or more of such infiltrations are to be seen stretching in towards the centre of the cornea, and although most commonly the course taken is a straight one throughout, there is sometimes a more or less decided bend made. When devascularised, a nebulous streak is left, which very slowly clears away, and is always most dense and persistent at the end farthest into the cornea. This portion often remains indeed a permanent opacity. The accompanying symptoms—circumcorneal injection, photophobia, and lachrymation—are the same, and exhibit much the same differences in intensity as in the case of phlyctenular keratitis. The affection is mostly strumous, and almost entirely confined to children. The *treatment* is the same as for phlyctenular keratitis, only in these cases Pagenstecher's ointment is even more valuable, and seldom fails to arrest the progress of the inflammation in a few days. It causes first of all a gradual disappearance of the band of vessels, and subsequently promotes absorption of the infiltrating cells. It is important to check the progress before the centre of the cornea is reached, as the opacity left is of course of much more serious consequence if situated directly in front of the pupil. In the rare cases in which the yellow oxide of mercury is not efficacious, an attempt may be made to interfere with the source of blood supply, by removing a piece of conjunctiva, or by cauterising the conjunctiva at the point where the vessels take their origin. This treatment is in general to be avoided, however. Where, at the time the case comes under observation, the portion of the infiltration at the end of the fasciculus of vessels should happen to occupy just the centre of the cornea, it is better to allow it to be pushed, as it were, a little further before trying to check it, so as to allow the pupillary area to be occupied by a vascularised portion, which is more completely absorbed, and therefore does not eventually interfere so much with vision.

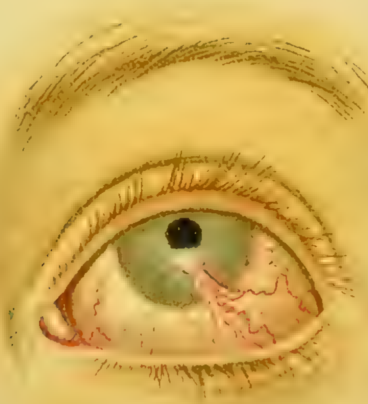


FIG. 34.—Fascicular keratitis.

PANNUS.—This consists for the most part of a development of new vessels in the superficial layers of the cornea. The vessels spring from those of the conjunctiva, and course immediately below as well as above Bowman's membrane. At the same time there is more or less irregularity of the corneal epithelium over the vascularised portion, and some diffuse infiltration into the tissues occupied by the newly formed vessels. There are great differences both in the extent and intensity of the vascularised area. When very dense the cornea assumes quite a raw fleshy appearance, a condition known, therefore, as *pannus crassus.*

Pannus occurs principally in connection with trachoma (see Fig. 35) and long-continued strumous infiltrations of the cornea. We may therefore distinguish a *trachomatous pannus* and a *strumous pannus.* Trachomatous pannus is apparently due to an extension of the granular inflammation from the conjunctiva to the cornea. In many cases, where the granulations are massive, there is found to be no pannus, while in others again, a marked degree of pannus is associated with comparatively slight alterations in the palpebral conjunctiva. This disproportion between the condition of the conjunctiva and cornea in different cases shows that the latter is not the result of friction, though this connection is often assumed from the fact that the

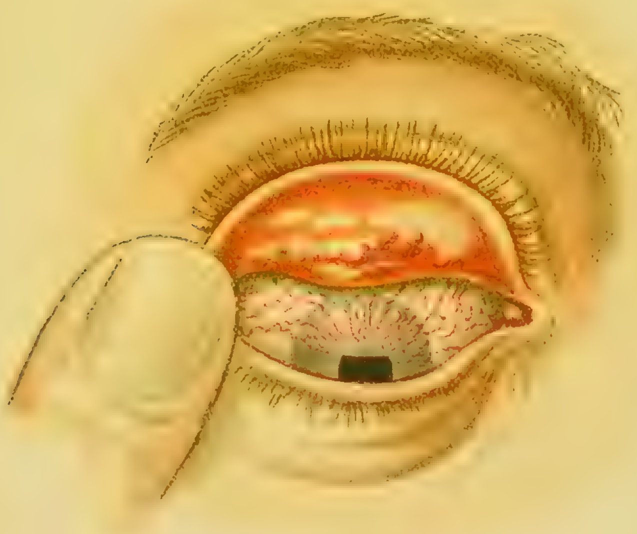


FIG. 35.—Old standing trachoma and pannus.

pannus often occupies only the upper portion of the cornea extending over an area corresponding to that covered by the upper lid. One frequently sees a pannus all at once set up, along with an acute exacerbation in the conjunctiva, in cases which have long remained free from corneal complication. In spring catarrh, too, in which the roughness of the conjunctival surface may attain a degree rarely if ever seen in trachoma, there is never any pannus. Strumous pannus is more frequent in young adults than in children. It is always a very chronic affection. Sometimes the infiltrations extend in depth, and become purulent, leading eventually to actual perforation of the cornea. The opacity produced by pannus may, in course of time, disappear under treatment, the vessels becoming first attenuated and less numerous. Long-continued cases often result in a hopeless opacity from a kind of cicatricial organisation of the infiltrated cells. Some again lead to alteration in the shape of the cornea, deepening of the anterior chamber, and eventually also to changes in the iris, with secondary glaucoma. An indication of the presence of such deeper complications is often afforded by a marked intolerance to the usual local applications. In other cases, perforation, followed usually by more or less shrinking of the eye, may take place.

The *treatment* of trachomatous pannus mainly consists in treating the conjunctival affection. The strumous form is best treated, unless it has gone on to ulceration, with Pagenstecher's ointment and massage in the shape of friction through the lid, along with suitable general treatment. Very long-continued dense cases of pannus may sometimes be caused to clear up by inducing a purulent inflammation of the conjunctiva. This may be done either by inoculating with pus from a case of gonorrhœal ophthalmia, or by bathing the eye several times with an infusion of jequirity. Both methods are somewhat heroic, as they are not without danger of producing very severe ulceration, perforation, and loss of the whole cornea. The jequirity inflammation was supposed, when first employed some years ago, to be capable of being modified at will by the strength of the infusion or the number of the applications. This has, however, proved to be impossible, and it is now rarely used. As to inoculation, it seems hardly justifiable to practise it, except in cases where the pannus exists in great density in both eyes.

Many cases are much benefited by the operation of *canthoplasty* for the enlargement of the lid aperture.

A form of extremely chronic and densely vascularised infiltration, not unlike pannus, is occasionally met with. The infiltration does not stretch for more than two to three mm. into the cornea, and is usually symmetrical. The etiology of this form of inflammation is not very clear. It is most commonly the upper margin of the cornea which becomes invaded, although sometimes the lower one as well. A dense nebula is left, and not infrequently a pretty regular astigmatism due to alterations in the curvature of the cornea. Cases which resist milder treatment are often improved by peritomy.

HYPOPYON KERATITIS.—A good many deep-seated abscesses of the cornea are associated with hypopyon, or a deposition of pus in the anterior chamber. Not infrequently this complication is met with in the more severe cases of strumous corneal inflammation, which occur mostly in children. Great differences exist in the density of the hypopyon; when very thick and fibrinous, it may not fall down to the bottom of the chamber, but remain sticking to the anterior surface of the cornea. Often, when this is the case, it is somewhat difficult to satisfy one's-self that the pus really lies in the chamber and not in the cornea. The name *onyx* has been applied to cases of supposed bagging of pus within the cornea below an abscess. Such a condition, though often described and figured in text-books, has no real existence. The appearance which has given rise to the term is always due to a deposition of pus in the anterior chamber, though from the fibrinous nature of the pus, it may not have fallen to the bottom of the chamber as is most common.

A very distinct clinical form of inflammation of the cornea is what is usually in this country specially understood by the term *hypopyon keratitis*, owing to its always being complicated with the appearance of pus in the anterior chamber. This affection occurs as an ulcer, one part of the irregular margin of which is densely infiltrated with pus. The characteristic feature of the ulcer is that it extends in the direction of this infiltrated margin, for which reason it has received the name, more expressive of its serpiginous nature, of *ulcus corneæ serpens* (see Fig. 36). The origin of this form of keratitis is in almost all cases a trauma, which has been followed by an inoculation of pus from a previously existing dacryocystitis or chronic conjunctivitis. Those whose occupations cause their eyes to be subjected to continual irritation, as well as occasional injuries, are most liable to contract this inflammation. Stonemasons, as well as farm labourers and colliers, are amongst the class of people in whom it is mostly found. It has now been definitely established that the inflammation is of a septic nature, and caused by the presence of germs which exist in the discharges from chronically inflamed mucous surfaces. As the ulcer extends, more and more pus becomes deposited in the anterior chamber. Although absorption of the pus no doubt constantly goes on, yet, as long as there is any active inflammation in the cornea, it is more rapidly formed than absorbed. Increase in the amount of the hypopyon is therefore a certain evidence of progress in the process of destruction in the cornea. As long too as the yellow margin of infiltration exists in the cornea, we may be sure that extension is taking place. The natural course of the inflammation is for the ulceration to go on spreading, until the whole of the cornea in the direction of the creeping infiltration has

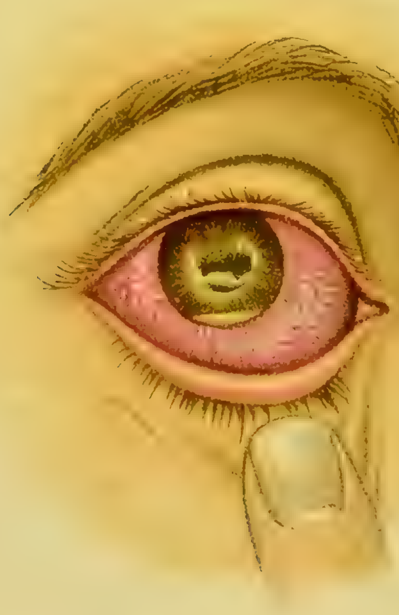


Fig. 36.—Hypopyon keratitis.

become involved, and as this often takes place in two or more directions, seldom in all at the same time, it is not an uncommon thing for the whole cornea to be ulcerated.

The exact origin of the pus in the anterior chamber is a disputed point, as there is often no apparent connection between it and the ulcer, but there can be no doubt, from the intimate connection which exists between the spreading of the ulceration and the amount of the hypopyon, that it is derived directly from the inflamed cornea; and in fact, in some cases, there is no difficulty, when it is more than usually fibrinous, in seeing it extending from the posterior surface of the cornea to the bottom of the chamber, and almost, as it were, trickling down. It is no doubt carried along the lymph channels, which appear to open into the chamber towards the centre of the posterior surface of the cornea. On oblique illumination, too, these channels can frequently be seen to be choked, and appear as fine radiating greyish striæ surrounding the portion of cornea involved. The complications of hypopyon keratitis are those already described as met with in connection with severe ulceration, though, owing to the greater tendency to superficial as compared with deep spreading, the more serious complications are not very frequent.

The *treatment* should be directed to first destroying the area of infiltration and then applying antiseptics. The most convenient, as well as the most efficient way in which the first indication is complied with, is to carefully burn down the marginal infiltration with the actual cautery, which was first recommended for this purpose by Martinache of San Francisco, either with the thermo- or galvano-cautery. When such is not at hand, the ulcer may be scraped down and then cauterised by applying a solution of nitrate of silver (10-20 grs. to ℥i.) directly to its surface. Both these operations can be performed painlessly with the aid of cocaine. Subsequently frequent bathing of the eye with a solution of corrosive sublimate (1 in 5000), followed by the smearing into the eye of an ointment of one part of iodoform to ten of vaseline, is very useful. If the cauterising be carefully performed—that is, if all the infiltrated margins be carefully burnt down—it is seldom that the process is not arrested, although it occasionally has to be repeated whenever any little infiltration makes it appearance. Those who do not succeed with this treatment fail from not cauterising sufficiently deeply, or from not repeating it on the first indication of any spreading of the infiltration. It is rare that a more severe but very successful treatment, which goes by the name of Saemisch's section, has to be resorted to. The method of performing this is described in the chapter on Operations.

If the ulcer has been central, as is often the case, it is necessary subsequently to perform iridectomy, so as to displace the pupil to opposite a clear portion of the cornea. In most cases it is advisable at the same time to treat the tear-sac in the manner already described. Owing to the tendency to iritis, it is well to keep the pupil dilated, until the healing process has advanced to such a stage that there is no further danger of such a complication.

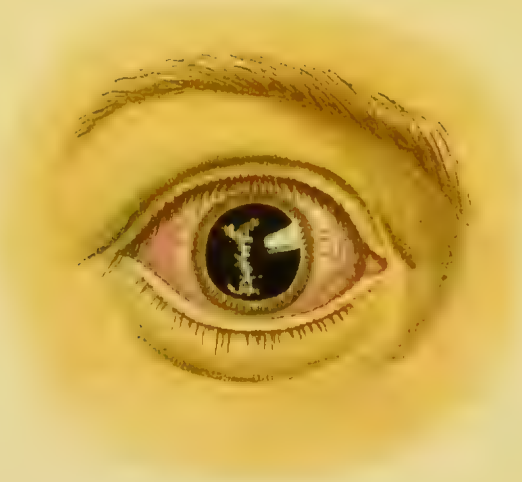


FIG. 37.—Dendriform keratitis.

DENDRIFORM (MYCOTIC) KERATITIS.—Another form of keratitis, which there is every reason to look upon as being also caused by some particular microbe, has only lately received attention since it was described by Hansen Grut. The characteristic of this inflammation is a great tendency to a kind of ramifying superficial extension (see Fig. 37). It is a very chronic affection, and the amount of infiltration surrounding the ulcerated rills which it forms is so slight as to render the peculiarity of its propagation liable to escape detection, unless a proper examination be made by oblique illumination. The pain caused by this inflammation is slight, though sufficient to give rise to some discomfort and photophobia.

When uncomplicated this form of keratitis is always superficial and not accompanied by hypopyon. In not a few cases, however, which begin in this way, and no doubt as the result of inoculation with more active micro-organisms, a serpiginous hypopyon ulcer may make its appearance. The cause is not known. It is a tolerably rare affection met with in both sexes, and for which the name dendriform superficial keratitis seems the most appropriate. The nebulous opacities, which remain for some time after recovery has taken place, have the characteristic ramifying appearance.

The *treatment* I have found most efficacious is to scrape the ulcerated rills with a small spud, such as is used for removing foreign bodies from the cornea, and afterwards apply a strong solution (1 in 1000) of corrosive sublimate directly to the cornea with a camel's hair brush, giving at the same time the iodoform ointment for frequent use. Latterly, I have used pyoctanin, which, though altogether useless in deeper corneal ulcerations, appears to be even more suitable than stronger antiseptics in dendritic keratitis. Chlorine water is also useful when freshly prepared.

SCLEROTISING KERATITIS.—Certain forms of marginal infiltrations, which extend into the cornea from the sclera, have the tendency to leave a dense white porcellaneous opacity in the cornea, which thus comes to resemble very closely the sclera in appearance. The margin of the cornea then looks as if it had been encroached upon by the sclera, and the name *sclerotising keratitis* very aptly describes the affection, so far as the inflammatory change in the cornea is concerned. The *treatment* is the same as for scleritis, as which it always originates. Nothing can be done for the clearing up of the dense opacity.

CLEAR CORNEAL ULCERS.—A clear form of ulceration of the cornea—that is to say, one in which neither the base nor the margins of the ulcer exhibit any infiltration—occurs almost exclusively in old enfeebled individuals, and is accompanied by little or no pericorneal injection, and often by few, if any, subjective symptoms at all, beyond more or less interference with vision according to its site. The infiltration stage of this type of ulcer is rapid, and therefore rarely observed; the healing stage is, on the other hand, very chronic. It is this circumstance which gives rise to the name *clear ulcer*. The *treatment* most likely to stimulate these ulcers to heal is the use of hot fomentations and eserine.

MARGINAL RING-SHAPED ULCER.—A long, narrow, ulcerated rill sometimes forms on the margin of the cornea, and extends in a ring-shaped manner round it. When occurring in children it is always caused by the confluence of a number of separate foci of inflammation. Such an origin, though probable, cannot always be demonstrated in the case of the less distinctly strumous form which is met with in adults. The infiltration is seldom great, but this form of ulceration is accompanied by a good deal of circumcorneal injection and other symptoms of irritation. By extending completely round, the clear central portion of the cornea may be deprived of its source of nourishment to such an extent as to necrose. In severe cases, which are fortunately rare, complete loss of the cornea may be caused by this form of ulceration. The etiology is not always clear, but it often comes on in connection with catarrhal conjunctivitis. The *treatment* should be directed towards preventing the spread of the ulcer. This is best done by paracentesis, which may be frequently repeated if necessary. Eserine and corrosive sublimate lotions are also useful. Occasionally it may be found advisable to use the actual cautery, followed by iodoform ointment.

VESICULAR KERATITIS.—A number of varieties, more or less distinct in their clinical aspects, occur, in which the surface of the cornea is raised up into vesicles, which may be small or large, and single or multiple. Sometimes, when small and multiple, the condition may be looked upon as a true herpes of the cornea. This form accompanies or follows a bronchitis or pneumonia, and there is, according to Horner, who has most accurately described it, a degree of anæsthesia of the cornea as well as diminution of intraocular tension at the same time.

One of the most interesting and distinct forms is that to which Hansen Grut has drawn attention—a *recurrent bullous keratitis*. This form only comes on after a superficial wound of considerable extent, and presents itself as a clear bulla, which cannot properly be seen except by oblique illumination, and which may occupy from one-eighth to one-half of the superficial extent of the cornea. The number of times that this condition may recur after a trauma, as well as the length of the interval between each attack, is subject to considerable difference. It is not uncommon to find a recurrence take place from three to six times a year for several years. The attacks invariably come on in the morning on waking, with circumcorneal injection, considerable, often intense, pain, and the sensation as if there were a foreign body in the eye. Under these circumstances, and with a previous history of an external abrasion of the cornea, followed by the same kind of pain, there is always to be found a corneal bulla. Sometimes, indeed, this cannot be seen without pressing the lid against the cornea, the epithelium of which is then thrown into a fold, or by catching hold of the anterior epithelial surface of the separated layer with an iris forceps, when it readily comes away. Only very rarely is the fluid contained in the bulla other than completely transparent. When yellowish or purulent some opacity is left, otherwise complete restitution takes place, so that the cornea becomes to all appearance absolutely healthy. A large superficial abrasion is very frequently followed by this form of inflammation, and, on the other hand, its occurrence at once leads one to the diagnosis, before the history of the original injury has been elicited. This, too, on account of the severity of the injury, there is very rarely any difficulty in obtaining. A cure takes place generally in from three to five days, except in the cases where the fluid of the bulla is discoloured. One of the most common causes of this affection is a scrape from a child's nail, and probably on this account, more than on any other, it is more frequently met with in women than in men. Other injuries, such as blows from branches or the end of an umbrella, not infrequently also, by causing a similar injury, set up the same train of symptoms. The cause of the tendency to recurrence appears to be imperfect healing. The epithelium which replaces that removed at the time of the accident is more liable to injury, so that after a time the slightest rub, which may take place during sleep, will cause it to be detached in the shape of a bulla. The *treatment* consists in removing the detached epithelium with a pair of iris forceps, and applying cocaine frequently to allay the pain, using at the same time some simple weak antiseptic lotion for bathing the eyes. There appears to be no way in which the recurrence can be prevented.

Another bullous form of keratitis, in which the bulla is not pellucid, occurs in cases of chronic inflammation of the eye, where there is or has been increase of intraocular tension. It is most frequently met with in old cases of glaucoma or irido-choroiditis, where the disease has led to complete, or almost complete, blindness. The bulla in these cases is due to an œdema of the cornea, which has led to the formation of a fibrinous false membrane between the epithelial cells and Bowman's membrane: it is this membrane, in addition to the epithelial cells, which forms the anterior wall of the vesicle. No treatment is called for in such cases, which are more of the nature of degenerative changes than of inflammation.

TUBERCULAR KERATITIS.—A rare disease of the cornea, which appears to be tubercular, consists in a dense infiltration, associated at the same time with the formation of a cheesy-looking mass in the anterior chamber. In most of the cases I have seen this infiltration has begun at the lower part of the cornea. No treatment is of any avail, and the eye is eventually lost by passing into a state of phthisis.

SECONDARY KERATITIS.

One form of secondary infiltration of the cornea is of very common occurrence, and is usually known as *diffuse interstitial* or *parenchymatous keratitis.* It never leads to any destruction of the corneal tissue. This disease produces a very characteristic appearance. It begins as a diffuse greyish and tolerably deep-seated opacity, which either stretches in from some portion of the periphery of the cornea, or, less frequently, first becomes evident in some more central portion. The whole cornea becomes pretty uniformly infiltrated, and at the same time a very definite stippling of the epithelial surface makes its appearance. Not infrequently different portions of the cornea are more densely infiltrated than others, or there may even be a very distinct punctiform arrangement of the infiltration, but there is never any prominence at such points as is seen in cases of primary infiltration. According to the density of the infiltration, the colour which it assumes is either white, grey, or yellow. When very dense it would be impossible, indeed, from the colour alone to say that no abscess was present. The degree of irritation is very variable, both in different cases as well as at different stages in the same case. There is usually, at some time or other, not a little circumcorneal injection, with photophobia and lachrymation, and often a more or less dense formation of new vessels in the cornea, giving rise to the appearance called the "salmon-coloured patch." This is well seen in Fig. 38. These vessels spring from the deep episcleral network which exists around the cornea, and therefore from branches of the anterior ciliary arteries. They can thus readily be distinguished from the newly-formed vessels of a pannus, which spring from superficial conjunctival trunks. As the opacity clears away, the new vessels, as well as the other symptoms of inflammation, gradually disappear. As a rule, the central portions of the cornea are the last to clear up. Both eyes are usually attacked by this inflammation, though rarely exactly at the same time; more commonly the disease is pretty far advanced in one eye before the symptoms make their appearance in the other.

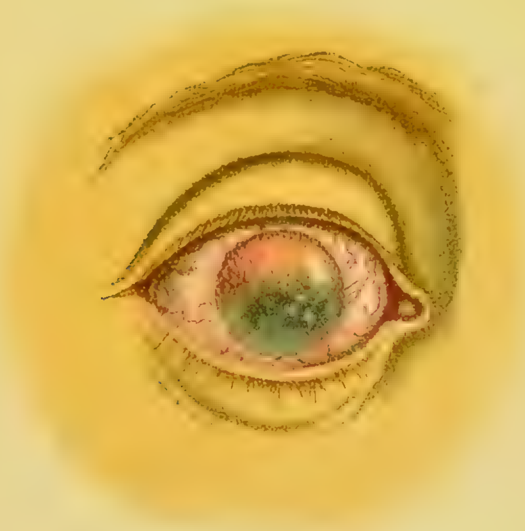


FIG. 38.—Vascularised interstitial keratitis.

Interstitial keratitis always lasts a long time—three to ten months as a rule. In some cases the duration is much longer.

There is a great tendency for the iris to become inflamed at the same time, and there are also observable in most cases changes in intraocular tension—at first generally an increase, but afterwards, and continuing far longer, a diminution, which is sometimes very considerable indeed. These circumstances, taken in connection with the absence of ulceration which characterises interstitial keratitis, sufficiently show that the disease is in reality not a true inflammation of the cornea itself, but merely an extension to it of inflammatory products derived from a focus of inflammation in the anterior portion of the uveal tract. They therefore justify the term secondary keratitis. The recognition of the secondary type, too, is of the utmost importance from a prognostic point of view.

The age at which the disease most frequently shows itself is from seven to twenty-one—mostly, perhaps, about puberty. Individuals who are of delicate constitution from any cause are subject to it. The most frequent predisposing cause appears to be inherited syphilis, as it is often met with in individuals in whom there exist other manifestations of this disease, such as scars round the mouth, prominent frontal eminences, &c.

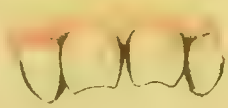


Fig. 39.—Hutchinson's Teeth.

A very common condition, too, in the subjects of secondary interstitial keratitis, is a peculiar state of the incisor teeth, to which attention was first called by Hutchinson, by whom it is also considered a manifestation of inherited syphilis. The cutting edge of the tooth, instead of being straight, is arched or notched in a more or less semicircular form, the sides being longer than the centre (see Fig. 39). Sometimes the form has not become definitely established at the time of examination, but there is often to be seen an indication that the cutting edge of the incisors is thinner than in the normal condition, and at the same time devoid of enamel. It is the wearing away of this portion which gives rise eventually to the characteristic notch.

Another complication is deafness, which appears in part due to a similar interstitial inflammation of the tympanic membrane, and in part to a periostitis within the channel for the auditory nerve, which probably causes the nerve to become involved in the inflammation.

In severe cases more diffuse choroiditis, as well as staphylomatous protusions of the anterior part of the choroid and the ciliary region of the eye, occur. The cases in which other manifestations of syphilis are evident appear to be those most frequently complicated by more deep-seated inflammations than such as give rise to the corneal opacity; cases, in short, in which, on that account, the prognosis is least favourable.

When the corneal opacity is very dense, so that little more than the movements of the hands can be made out, on account of the optical interference produced by that opacity alone, it may sometimes not be very easy to ascertain whether or not any serious complication exists. As a general rule, it is not difficult, after a little practice, to satisfy one's-self as to whether the visual acuity answers to the opacity or not. Even the very densest corneal infiltration produced by this disease should permit of the movements of the hand being seen close to the eye, so that if vision is further reduced, to, for instance, mere perception of light, there is certainly some complication present. In uncomplicated cases, too, the field of vision is of course of normal extent.

In a large majority of cases of interstitial keratitis the opacity eventually clears up entirely, or leaves but the faintest trace, discernible only on oblique illumination. The *prognosis* depends in fact on the severity of the primary affection. When the iritis is slight, and no choroiditis or staphylomatous protrusions make their appearance, it is good, even though the opacity should be at one time extremely dense. Occasionally cases beginning as interstitial keratitis go on slowly to shrinking of the eye. Sometimes two or more attacks may occur in the same individual at considerable intervals of time.

The disease runs a definite course, and it is very doubtful, just as in the case of other deeper uveal affections, in how far any *treatment* is of any avail. The eyes should certainly be protected from strong light, and the patient not allowed to read. When there is much circumcorneal injection the pupil should be kept dilated with atropine, so as to avoid the worst consequences of a complication with iritis. Attention to the skin and to the health generally, moderate exercise, and the use of syrup of the iodide of iron internally, either alone or combined with a little iodide of potassium, are indicated. In the distinctly syphilitic cases mercurial preparations may be tried. When ciliary staphylomata develop, it is advisable to try eserine, which appears to have some influence in preventing their extension. All surgical interference, even such a simple measure as paracentesis, should be avoided, except in cases where there is a marked shallowing of the anterior chamber. In these iridectomy should be performed as soon as possible.

Other less characteristic and altogether much less frequent forms of secondary keratitis are met with, in connection usually with old-standing cases of choroiditis and irido-choroiditis. In these the prognosis is relatively much worse than in what is usually known as interstitial keratitis, owing no doubt to there being less tendency towards the healing of the primary process on which they depend.

Occasionally a peculiar form of disease is met with, which at first looks very like a secondary interstitial keratitis, but which afterwards becomes associated with a tubercular-looking deposit in the anterior chamber and iris, as well as a dense whitish opacity in the corresponding portion of the cornea. The affection is extremely chronic, and appears to be of a tubercular nature, and allied to, though by no means the same affection as, granuloma of the iris. I have usually seen it in young adults.

NEURO-PARALYTIC KERATITIS.—When the fifth nerve is paralysed there is a tendency for the cornea to become inflamed. Different forms of inflammation may occur under such circumstances, which all, however, exhibit, besides the accompanying anæsthesia, a marked slowness in healing, if indeed they do not lead to the total destruction of the cornea.

This form of keratitis has been often produced experimentally in animals, and various views as to its etiology have been from time to time put forward. It has been looked upon as due entirely to interference with the trophic supply to the cornea. This view was originated by Majendie, and was that held by von Graefe. Many subsequent experimenters believed the keratitis to be always started by injury, but admitted that the power of resisting such injuries was reduced by the trigeminus lesion. Others again, and notably Snellen, have denied that the inflammation has anything to do with a severance of trophic supply. It is indeed most probable that the main cause of so-called neuro-paralytic keratitis is the greater liability of the cornea to traumata, and to small abrasions from the more rapid evaporation and insensibility which paralysis of the fifth produces. The latest experiments, such as those of von Gudden, Böckmann, and von Hippel, have pretty clearly shown that the trophic hypotheses are untenable. Possibly, as has been suggested by Eberth, the keratitis which, in common with other experimenters, he found to arise so readily in animals in which the fifth nerve has been divided, is in reality a mycotic one.

The *prognosis* in all cases of neuro-paralytic keratitis is necessarily bad.

The *treatment* consists in as far as possible protecting the eye from external influences, by keeping it tied up, and frequently applying antiseptic lotions. The corneal changes which are met with in herpes zoster frontalis may be looked upon as neuro-paralytic in their nature. In some cases a number of small herpetic blebs form on the cornea, very similar to those met with on the skin. These burst and leave infiltrated margins, and eventually more or less opacity. In other cases the first appearance in the cornea is of one or more infiltrations or ulcerations. The affected portions of the cornea are generally anæsthetic, and often remain so, long after healing has taken place. A corneal complication appears to be met with in about one-fourth of all the cases of herpes zoster frontalis. According to Hutchinson, the cornea is only affected when an eruption at the side of the nose indicates a participation of the naso-ciliary branch in the affection. This connection is certainly frequent, and in my own experience it has been invariable; but cases are on record where the cornea has been affected without the skin of the nose being implicated, while at the same time the skin of the nose has been found to be affected and not the cornea. Great pain usually accompanies, as well as sometimes precedes and follows, the herpetic inflammation on the cornea, just as is the case with similar manifestations elsewhere. No very efficacious treatment is known for this affection. Sometimes the pain is relieved by tying up the eyes, in other cases nothing except morphia gives relief.

NON-INFLAMMATORY AND DEGENERATIVE CHANGES IN THE CORNEA.

Transverse calcareous film.—A very curious form of corneal opacity is met with, mostly in eyes which are at the same time the site of some old-standing inflammatory changes, such as chronic iritis with occlusion of the pupil, and it may be, with calcareous degeneration of a cataractous lens. This consists of a transverse film running across the cornea always at that part which corresponds to the half-closed lids. It generally begins at the corneal margins, and spreads from both sides towards the centre. The breadth of the band is tolerably uniform throughout, being in different cases from about $\frac{1}{10}$ to $\frac{1}{6}$ inch. The opacity is finely punctiform, and varies in colour from a grey to a pretty pronounced yellowish-brown. Examined with a magnifying-glass, it is found to be composed of a number of granules, which are very regularly distributed and of much the same size throughout. Its edges are very sharply defined, and do not anywhere merge into the healthy cornea. It is often least dense at the centre of the cornea over the pupil, but altogether much more intransparent than its appearance at any

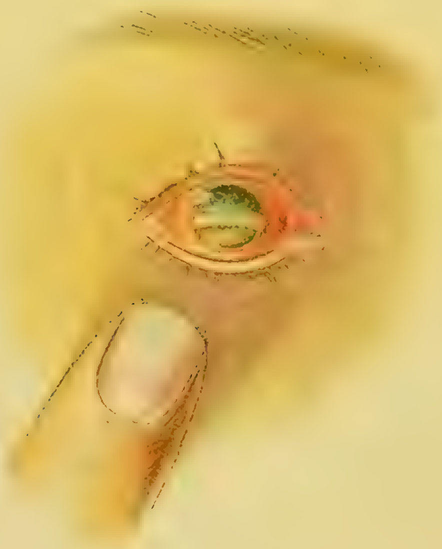


Fig. 40.—Transverse calcareous film of cornea (in a shrunken eye).

distance would lead one to suppose, owing probably to the calcareous degeneration which takes place in the deposited matter. There is rarely any disturbance of epithelium above the opacity.

This form of opacity almost invariably occurs in both eyes, though it may exist to a much more marked extent in the one than the other at the time of examination. It is met with at all ages and in both sexes, though more frequently in men. The opacity forms very slowly, and then remains for an indefinite time stationary. The surrounding cornea is as a rule perfectly healthy, but there is a variety of the same affection which makes its appearance in old-standing leucomata. From the fact that some form of uveitis is almost always found to be associated with it, it seems probable that, even when the disease is apparently primarily a corneal one, there is nevertheless some deep-seated affection, which, though it may escape observation at the time, afterwards develops into something more manifest.

Glaucoma is often met with, sooner or later, in connection with these calcareous films in the cornea, and for this reason it seems advisable, as was recommended by von Graefe, to perform iridectomy early. Any other *treatment,* such as scraping away the film or removing it by means of very dilute nitric acid, as has been proposed by Nettleship, is of very little permanent use, as it again develops.

Cases which have been examined microscopically have all been found to be caused by a formation of chalky matter in Bowman's membrane and the immediately subjacent parts of the cornea. How the chalk comes to be deposited there, and why it should just occupy that portion of the cornea which is most uncovered by the lids, has not yet been explained. The disease seems first to have been described by Dixon, but did not attract much attention until long afterwards, when von Graefe pointed out its connection with glaucoma, a connection which, however, does not appear to be as constant as von Graefe believed.

ARCUS SENILIS.—This is a whitish and generally completely circular opacity, which extends round the margin of the cornea in old people. It begins at the upper and lower margins of the cornea, and varies considerably in thickness in different individuals as well as often at different parts of the cornea. Very often the arcus is separated from the scleral margin by a clear or less opaque strip of cornea. The appearance is due to fatty degeneration of corneal cells. Wounds through this area of degeneration heal much as in the normal cornea.

Conical Cornea.—Owing to a thinning of the central portion of the cornea, a change of its curvature may take place, whereby the normal ellipsoidal form becomes converted into one more resembling a hyperboloid of revolution. The curvature of the central portion becomes greater, that of the peripheral portions less than normal (see Fig. 41). This gives rise to myopia, to a degree of irregular astigmatism with polyopia, and, in the most pronounced cases, to some diminution in the field of vision. The apex of the conical cornea always lies slightly below the centre, and is often more or less opaque. It may be so thin that it can be seen with the ophthalmoscope to pulsate synchronously with the pulse. This phenomenon was first observed by Javal, and has lately been confirmed and demonstrated by Gullstrand. The condition is almost invariably bilateral, though frequently not developed to the same extent in both eyes. The pathology is not properly understood. It is not known what causes the thinning and want of resistance in the cornea. It appears to be equally frequent in women and men; according to some, women are more subject to it. The conical bulging begins generally after puberty, in the early years of adult life. It is not improbable that the degenerative change in the cornea may go on for some time before the bulging begins. There is a decided tendency

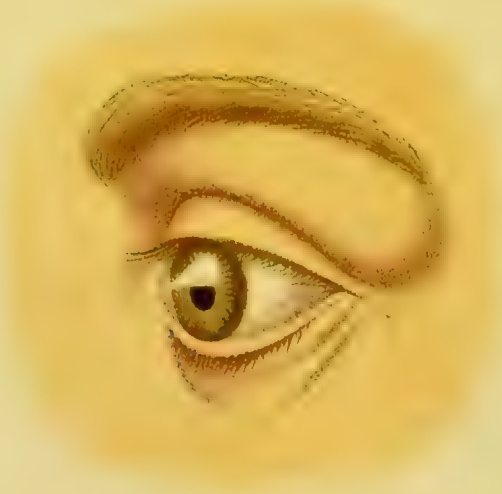


Fig. 41.—Conical cornea.

for the condition to remain stationary after having progressed for some time. I have seen several cases where the condition has lasted for upwards of thirty years without destroying sight, or indeed rendering reading altogether impossible. Not infrequently however the protrusion gradually increases more and more, and is associated with more and more opacity.

The *diagnosis* of conical cornea is made without difficulty by mere inspection when the protrusion is pronounced. The conical shape can be readily seen by looking at the cornea from the side. When slight, or only beginning, it is diagnosed by means of the ophthalmoscope or Placido's disc. With the ophthalmoscope a peculiar circular-shaped shadow is thrown on the fundus, which alters its position with the movement of the mirror. It corresponds to a zone through which fewer rays pass to the observer's eye, owing to the rays on one side of the zone being convergent and on the other divergent as they emerge from the eye. With Placido's disc the great diminution in the size of the image of the rings on the apex, and their elliptical or hyperbolic distortion in the vicinity of the apex, is very characteristic.

Treatment.—Conical cornea admits generally of some, though rarely of very complete, optical correction. The higher numbers of negative spherical and cylindrical lenses, either separately or combined, are those which are most likely to prove of service. Sometimes a stenopaic aperture or slit may be added with advantage. With Raehlmann's hyperbolic lenses, manufactured in Rathenow, a much greater improvement can occasionally be got than by any other optical means. They may, too, be combined with convex spherical glasses. The disadvantage of the hyperbolic lenses is that they only correct when their apices coincide with those of the corneal cones, which is only the case for one position of the eye. Their use as spectacles, at any rate, must therefore be limited. In many cases the possible optical correction is so small that some operation is indicated. An operation may in any case be attempted when there is opacity of the apex of the cornea. All operations which have been devised for conical cornea are more or less doubtful in their results. That most likely to prove of benefit is the removal of an elliptical portion from the apex, or from near the apex of the cone. Trephining, as recommended by Bowman, is undoubtedly of use sometimes. The same may be said of von Graefe's treatment,—cauterising of the apex, and subsequent iridectomy. Displacement of the pupil without iridectomy (iridesis) has also been tried. It must be remembered that such operations are not altogether free from danger. Suppuration of the cornea, followed it may be by suppuration of the whole eye—panophthalmitis—is occasionally set up.

Another form of misshapen cornea is what is called *keratoglobus.* In this condition, which is not nearly so common as conical cornea, and probably always congenital, there is a more general thinning, and consequently a more globular distension,

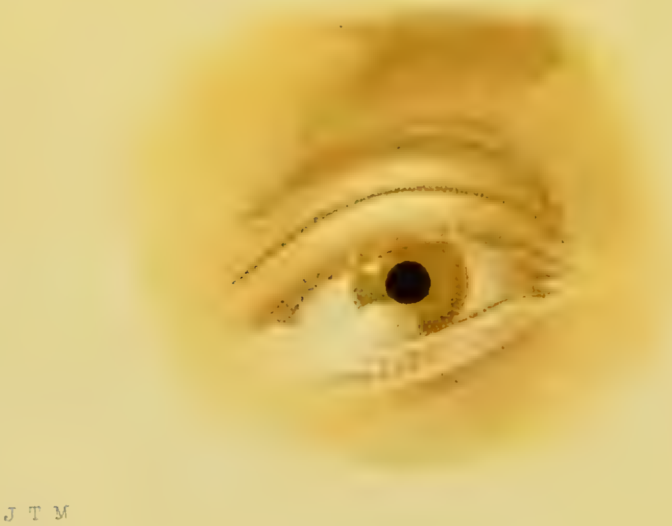


FIG. 42.—Dermoid cyst of cornea.

of the cornea. Examined with a Placido's keratoscope, there is little or no distortion of the circular rings. The cornea is sometimes so thin, that when paracentesis is performed it falls in, crumpled up like paper. In the higher degrees the condition has often existed for a long time; the cornea may become somewhat opaque all over, and the disease is liable sooner or later to lead to secondary glaucoma. No treatment is likely to be of much avail. Iridectomy has been found to be dangerous, as liable to lead to loss of vitreous and purulent cyclitis. It should therefore be deferred until glaucomatous symptoms have made their appearance.

TUMOURS OF THE CORNEA.—*Dermoid Cyst.*—This may be found as a congenital growth at any part of the margin of the cornea. It seems usually to remain of much the same size during life, though occasionally found to increase after birth. The cyst is firmly attached to the cornea by half, or rather more than half, of its base, the other portion being attached to the episcleral connective tissue. A dermoid cyst of the cornea, owing to its position and size, seldom interferes with vision, but may require removal on account of producing more or less conjunctival irritation, or on account of its unsightly appearance.

Fibroma of the Cornea.—This is an exceedingly rare affection. It occurs as a flat growth of densely white appearance, involving the superficial portion of the cornea, over the whole of which it very slowly spreads. When met with before it has involved the whole cornea, it should be removed by slicing off layer after layer, until the transparent tissue below it is reached.

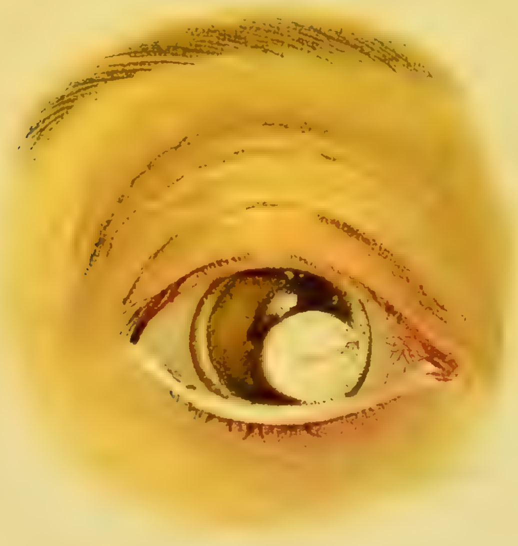


FIG. 43.—Fibroma of cornea.

Malignant Tumours of the Cornea probably never occur primarily. They either involve the cornea by extension from the deeper tissues of the eye (ciliary body, choroid, &c.), or from the conjunctiva, in which case only the superficial layers of the cornea are implicated. The principal forms met with are sarcoma, generally of the melanotic type, and epithelioma. Owing to their loose attachment to the cornea, they may easily be scraped off, but the tendency to recur usually necessitates removal of the eye.

CONGENITAL MALFORMATIONS OF THE CORNEA are rare, unless we include under such corneal astigmatism. Sometimes the cornea is much smaller than normal, a condition known as *microcornea*; in others it is much too large—*macrocornea*. Both these deviations are most frequently accompaniments of more general deformities—*microphthalmos* and *macrophthalmos* respectively—though they may exist alone, or at all events constitute the most evident deformity. A congenital opacity of the cornea, either partial or complete, is also met with very rarely.

INJURIES TO THE CORNEA.—Superficial abrasions of the cornea, which rarely involve more than the epithelium, are frequently met with. An injury of this nature may result from a scratch from the finger-nail, or from the eye being struck with some object—*e.g.*, the branch of a tree, end of an umbrella, &c.—or from the rubbing of some small foreign body which has remained for some time in the conjunctival sac. The corneal abrasion, owing to the irritation of the numerous delicate nerve fibrils which it causes, always gives rise, and more especially when the eye is not kept closed, to very considerable pain, often accompanied at the same time by the sensation as if a foreign body were present in the eye. When the abrasion is large it may give rise to a form of recurrent bullous keratitis (see page 135). The pain is at once allayed by cocaine, and the *treatment* consists in using frequently some mild antiseptic wash, so as to avert any possible subsequent inoculation of the wounded cornea. The extent of a superficial abrasion of the cornea may be made very apparent by dropping a drop or two of a half per cent. solution of fluorescine into the conjunctival sac, and washing away the excess with water. The area of the abrasion is thereby coloured a vivid green, while the rest of the cornea on which the epithelium is intact remains uncoloured. This method of staining was introduced by Straub some years ago. I have constantly used it since then for demonstrating abrasions and superficial ulcerations, such as dendritic keratitis, to students.

Penetrating wounds of the cornea of all sizes are caused either by sharp instruments, or by severe blows with blunt objects, which lead to a bursting of the eye. The *prognosis* in such cases depends greatly upon the nature of the injury, and upon the complications. A mere cut of the cornea, if made by a sharp, clean instrument, and if not very extensive, always heals by first intention. If not directly opposite the pupil, it may result in little or no interference with vision. One circumstance not unlikely to happen, and which may call for surgical interference, is a prolapse of a portion of iris through the wound. When this happens, and the case is seen shortly after the accident, the eye should be bathed well with corrosive sublimate solution, and the iris, if not much bruised, returned by careful manipulation with a blunt instrument (preferably one made of gutta-percha). When the prolapsed iris is allowed to remain in the wound, healing by first intention cannot take place, and the curvature of the cornea is more or less altered ; besides, there is a danger of iritis being set up. If the iris has been at the same time bruised, and the case has only come under observation some days after the accident, the prolapsed portion should not be replaced, but removed with the iris forceps and scissors. The great danger in such wounds is the possibility of septic matter having been introduced into the eye by the instrument with which the accident was caused. When this has been the case, inflammatory reaction, which not only interferes with the healing of the corneal wound, but which may lead to more deep-seated destruction, is liable to occur.

Small particles of dust, coal, or metal, and husks of grain, are very liable to become embedded in the cornea. The distance to which they enter depends on their size and shape, and the force with which they are driven against the eye. In most cases the foreign body lies superficially in the corneal epithelium. In the case of small portions of metal it very often happens that the cornea is not only wounded, but at the same time slightly burnt, owing to the piece of metal being hot at the time it strikes the eye. This, which is popularly called a " fire," differs from other foreign bodies by being as a rule less firmly embedded, owing to the destruction of the immediately surrounding tissue. A brownish ring is often left, too, after their removal, partly an eschar and partly rust, which, if not scraped away, is some days in falling off. Usually a foreign body gives rise to sudden pain when it lodges in the cornea, and often more or less pain remains until it is removed. The pain is more especially complained of on movement of the lids over the cornea. Except, too, in the case of smooth and chemically non-irritating substances, the eye becomes rapidly injected, and the patient suffers from photophobia and lachrymation. Husks of grain become embedded with their concave surfaces towards the cornea, and the lid passing over their smooth surfaces does not give rise to much pain. The method of removing foreign bodies from the cornea is described in Chapter XVIII.

SCLERO-CORNEAL RUPTURES.—When the coats of the eye are ruptured by a severe blow, the line of rupture is most frequently roughly concentric with the margin of the cornea, and from one to two millimetres distant from it. The eye, in fact, gives way close to the true corneo-scleral junction at the angle of the anterior chamber. Such ruptures are usually in the upper portion of the corneo-scleral junction, but may be in any part of it. They rarely if ever involve the ciliary body. When large they may permit of the escape of the lens, and it is in this way that a subconjunctival dislocation of the lens takes place. The vitreous, too, and even the retina, may be propelled through an extensive rupture of this nature. A less extensive wound generally gives rise to quite a characteristic appearance. A portion of the iris becomes prolapsed into it, so that the centre of the pupil is drawn to the wound, and an apparent coloboma results. The conjunctiva which remains intact is often raised, too, in the form of a bleb over the wound by aqueous humor infiltrated below it, and the escape of aqueous in this manner leads to a marked shallowing or abolition of the anterior chamber.

Owing, no doubt, to the integrity of the conjunctiva as a general rule, corneo-scleral ruptures are rarely followed by severe inflammation. On the other hand, they seldom heal without leaving a great deformity. This is due to the separation of the lips of the wound by prolapsed iris, &c. On this account they usually call for operative *treatment*, which should not be undertaken without the usual antiseptic precautions. In fresh cases an opening should be made in the conjunctiva a quarter of an inch or so behind the rupture, and parallel with it. The conjunctiva should then be undermined, so as to form a flap, which can be lifted away from the wound. When this has been done, the prolapsed iris is removed as completely as possible, so as to admit of the edges of the wound coming in contact. The conjunctiva can then be carefully stitched over the wound. There is rarely any use trying to replace the prolapsed iris, as it is generally lacerated as well. In cases which have been allowed to heal after the accident, and which have resulted in great deformity and corresponding interference with vision, a method of operating advocated by Nuel would seem to offer the best chance of improvement (see Chapter XVIII.).

Deposits in the Cornea.—Some metallic salts used in solution as lotions for the eye may leave deposits in the cornea. This danger is greatest with lead lotions, which should never be used where the cornea is inflamed. A white deposit of lead is very rapidly formed on the surface of a corneal ulcer, and may often occasion much greater defect of vision than would arise from simple cicatrisation. As such lead deposits are generally superficial, more or less improvement may result from scraping them away. Nitrate of silver in the same way may lead to an opacity, but this is not nearly so unsightly, being a deposit of a number of more or less closely packed brownish or black dots. A much more prolonged use of this substance is required, too, to produce much staining, than is the case with lead lotions.

CHAPTER V.

DISEASES OF THE CRYSTALLINE LENS.

ANATOMY.—Several changes take place in the lens during life. In the young it is perfectly colourless; later on in life it is yellowish. The change in *colour* is accompanied by an alteration in *consistency*, bringing with it a diminution of elasticity, which is the cause of the gradually diminishing amplitude of accommodation met with as age advances. The *shape* of the lens is much more globular in infancy and childhood; the increase which takes place is therefore mainly at the equator. In this way, too, the dioptric value of the lens accommodates itself to the change which takes place in the length of the eye as it increases in size.

The *growth* of the lens has been shown by Priestley Smith to take place throughout life, and not to cease with the attainment of adult life. From 25 to 65 its weight and volume are increased by one-half, and its diameter by one-tenth.

The explanation given by Priestley Smith of this fact, which is not without importance with reference to possible disease in advanced life (see Chapter on Sympathetic Ophthalmitis) is as follows:—"The lens is derived from the cuticular epiblast, and its mode of growth is analogous to the cuticle. But its cells, unlike those of the cuticle, are not cast off as they grow old; they are laid down layer upon layer within a closed capsule, the younger fibres surrounding the older. In consequence of this unique arrangement, and in spite of the shrinking of the older cells which form the nucleus, the growth of the lens does not cease with that of the rest of the body, but is continuous unless some morbid process intervene, throughout the whole period of life."

Numerous measurements which Priestley Smith has made give to the lens an altogether greater thickness than it is generally supposed to have. The fibres towards the centre of the lens are more compressed than the superficial ones, and contain less water, but never any keratine. The lamellar arrangement of the lens fibres can be seen by macerating, by which means a separation of the different lamellæ takes place sooner than a dissociation of the individual fibres, the cementing substance being present in greater quantity between the edges of the fibres than between their surfaces. There is a gradual increase in the *refractive index* of the different lamellæ passing from the surface to the centre of the lens. This difference becomes less marked as age advances by the gradual development of a hard central portion, the so-called *nucleus,* which becomes apparent about the time, or shortly after the period of full growth, and slowly invades the more superficial portions of the lens, the refractive index of which, corresponding to the greater density, is increased. The refractive index of the superficial fibres of the lens differs but slightly in the eyes of young individuals from that of the aqueous humor. The increase in refracting power, which comes on with age, brings about, however, a considerable difference between that of the lens and aqueous where they meet, and the reflection of light from the surface of the lens in old people, which this abrupt change in refractive power admits of, is the cause of the greyish and semi-opaque appearance of the pupil which is so frequently observed.

The capsule of the lens is a highly elastic, and to all appearance homogeneous membrane, which attains its greatest thickness at the anterior portion. While the lens fibres are known to be histogenetically epithelial, it still remains doubtful whether the capsule is originally an epithelial or a connective tissue. The latest investigations indeed seem to point to a double origin. The anterior portion of the capsule—what is usually spoken of as the *anterior capsule*—is covered on its internal surface by a single layer of epithelial cells, which play an important part in the physiology of the lens.

The normal *position* of the lens is that in which it is suspended at an equal distance all round from the circular ciliary body, so that its axis is nearly, if not absolutely, centred with that of the cornea. The centre of the anterior surface lies in the same plane as the iris, while the equator, owing to the curvature of the surface, is considerably behind that plane. A space is therefore left between the posterior surface of the iris and the anterior surface of the lens. This is the so-called *posterior aqueous chamber.* The suspensory ligament of the lens, or *zonule of Zinn*, which is a differentiation of the hyaline membrane of the vitreous, has an extensive attachment to the processes of the ciliary body. It does not, however, follow all the depressions in these processes, but is firmly attached to the elevations. Spaces—the so-called recesses of the posterior aqueous chamber—are thus left between it and the ciliary processes. It is at its other end closely fused with the lens capsule, more particularly the anterior capsule. The intermediate free portion of the membrane fills in the *perilenticular space.* This portion appears to be narrower the older the individual. Behind the zonule of Zinn, and between it and the vitreous, or more correctly between the fibres of the zonule which pass to the anterior and those which pass to the posterior capsule, is a space called the *canal of Petit*, which is of importance in connection with the nourishment of the lens. The intimate structural as well as anatomical connections which exist between the suspensory ligament and the vitreous and anterior portion of the uveal tract, render a participation of that structure in deep-seated diseases of the eye not infrequent.

CATARACT.—When the lens, which in its physiological state is transparent, becomes opaque, there is said to be cataract. The opacity may involve the whole or only a portion or portions of the lens, *i.e.*, the cataract may be *complete or partial.*

Anatomically, we may distinguish two forms of cataract—(1.) *Lenticular* cataract; (2.) *Capsular* cataract. The first form is a result of the opacity of the lens fibres or true lens substance, the second of the cells lining the capsule of the lens. The two forms may exist together or separately.

Clinically, cases of cataract may be grouped with advantage under the following heads:—(1.) *Senile* cataract, in which the opacity is due to an idiopathic degenerative change in the lens substance, unconnected necessarily with any other condition which leads to an impairment of the functions of the eye. According to the extent as well as the nature of this degenerative change, different clinical sub-groups have to be distinguished. (2.) *Juvenile* and *congenital* cataract, of which there are also various forms. (3.) *Complicated* or *secondary* cataract, in which the opacity of the lens is the result of more or less destructive changes in the deeper parts of the eye. (4.) *Traumatic* cataract;

and (5.) *After-cataract,* or the opacity which remains or becomes developed subsequently to the removal, by artificial or natural means, of the opaque lens.

The transparency of the lens is only maintained when no great change takes place in its nutrition. A normal nutrition requires, as a rule, a normal position of the lens, and always integrity of its capsule. The main cause of opacity appears to be a too rapid or irregular abstraction of fluid, which is favoured by some interference with the supply, or some abnormality in the reconstitution of the nutritive fluid.

It is only in eyes where no nucleus has yet formed in the lens, and therefore in the young, that the whole substance of the lens is capable of undergoing a degenerative change which leads to soft cataract. The nucleus may lose much of its transparency from sclerosis and increasing coloration, but it is not the same process of degeneration as takes place in the less altered portion of the lens.

Senile cataracts are of two kinds—*cortical* and *nuclear*. The former is much the more common. In this form only that portion of the lens which surrounds the nucleus becomes opaque. The colour of such cataracts, when the whole of this portion (the cortex of the lens) has become opaque, depends on the size and colour of the sclerosed nucleus, as well as on the degree of opacity of the cortical layers. When the opacity is dense and the nucleus small, a very uniform white appearance is met with; on the other hand, a greyish, and often yellowish tinge, is given to the cataract by the shining through of a large nucleus. In the first case, the cataract is comparatively *soft;* in the second, it is what is called *hard.* As a general rule, it may be taken that the older the individual is before the cataract begins, the larger is the sclerosed central portion, and the smaller the cortical opacity.

By observing carefully the colour of a cataract, which can only be done by dilating the pupil well, a fair idea can be formed as to its consistency. Such an examination should be made a matter of routine practice, as the result of it may occasionally influence the choice of operation. A comparatively soft senile cataract can be extracted through a smaller incision into the eye than a hard one, and at the same time cause less bruising to the iris, but too much confidence should not be placed on the diagnosis as to consistence; moreover, when the incision is made in the corneo-scleral margin, and antiseptic precautions taken, the necessity for making it as small as possible is not so great as in the case of a corneal section, which is not much practised now. The colour of the opaque lens may be bluish, white, or more or less yellowish, and even brown. Bluish cataracts in young people have generally also a mother-of-pearl-like sheen, and are always soft and swollen. A similar appearance in older individuals indicates softness of the cortical matter, but the existence at the same time of a hard nucleus, corresponding to the age of the patient, must not be forgotten. White cataracts may be either hard or soft; they are hard when distinctly striated, soft when homogeneous or merely spotted. Yellowish or brownish cataracts are hard.

An abnormal degree of sclerosis may involve the whole lens, which in such cases is smaller than normal. This only occurs, as a rule, in very old people, and comes on very slowly. This form, in which no true cortical substance remains to undergo degenerative changes, and yet the sclerosed portion becomes greatly deprived of its transparency, is what may be called *nuclear senile cataract*. Often the lens is very darkly coloured, when the cataract is said to be "black." Nuclear cataract is therefore essentially a hard cataract.

Ordinary senile cataract, where there is a certain, though varying, proportion of cortical substance, begins apparently as the result of some irregularity in the process of sclerosis of the central portion. According to Priestley Smith, a decrease in size and weight of the whole lens precedes the formation of cataract. The first signs are to be seen in the region between nucleus and cortex. Variously shaped opacities, mostly radiating lines or striæ, which appear white by reflected and black by transmitted light, occur here: these gradually become confluent, and extend towards the capsule. This change is often associated with considerable increase in volume, so that the iris is pushed forwards and the anterior chamber shallowed.

When the whole cortex has become opaque, the cataract is said to be *mature* or *ripe*. The term has reference to its then being capable of being shelled out of its capsule, to which it is otherwise somewhat adherent. There is considerable difference however, in ripeness taken in this sense, as in some cases, even when the opacity extends right up to the capsule, the adhesion between lens substance and capsule is pretty firm, whereas in other cases again a semi-opaque lens separates very completely and readily from the capsule. It is not as a rule difficult to make out if the opacity reaches to the capsule or not; where there is any doubt, the diagnosis may be assisted by observing whether, when a light is held to one side of the cornea, the portion of the iris to the same side throws any shadow on the opaque lens; if it does, there must be an intervening portion of transparent lens. It must not be forgotten that a similar shadow may be seen in cases of over-ripe cataract. These cases may be diagnosed by observing that the anterior surface is at the same time flattened, whereas the unripe cataract presents a distinctly curved surface. Tremulousness of the iris—iridodonesis, is also frequently met with, where the cataract is over-ripe, and has undergone retrogressive changes.

After the condition of ripeness has existed for a longer or shorter time, further changes may take place, which are characterised as stages of over-ripeness. The opaque cortical substance may to some extent shrivel from loss of fluid, and in this way a little of the sight—as a rule, only a very little—be regained. A little trembling of the iris with the movements of the eye is often observable. Usually, too, dense white points or irregular lines are to be seen in the lens. On the other hand, and much less frequently, the cortex may become more fluid, to such an extent, indeed, that the nucleus moves about in it, and gravitates to the lowest point of the lens. Such fluid cataracts are called *Morgagnian*. The Morgagnian cataract is easily distinguished, too, by the position of the yellow nucleus which has sunk to the bottom of the opacity. The diagnosis may often be made without dilating the pupil, by observing the difference in colour of the opacity when the patient is standing up or lying down. In the former case it is generally yellowish; in the latter milky white.

The time which elapses before a senile cataract becomes mature varies within very wide limits. It is impossible in any particular case, as a rule, to predict even approximately how long the existing opacity will take to become complete. No regularity is shown in the process, which at times advances more rapidly than at others. Some cases, for instance, rapidly advance till a considerable degree of opacity is reached, and then only progress slowly to maturity, while others advance slowly at first, and, after reaching a certain stage, very quickly end in complete opacity. Probably, however, no cases remain absolutely stationary when once a separation has taken place between the nucleus and cortex, and striæ are seen beginning in the region between them. In cases where small opacities are found in other situations, there does appear to be some possibility of the condition remaining stationary—at all events for some considerable time.

The degree of blindness to which the progressing cataract gives rise depends a good deal on the site of the intransparent portions of the lens. If, for instance, as often happens, the cortical substance in the pupillary area is more affected than elsewhere, the vision is more seriously interfered with than in the case where the same degree of opacity exists at another part of the cortex. In such cases vision is better in the shade than when the pupil is more contracted by a strong light, and a temporary benefit may be got by the use of a weak mydriatic. Less frequently, owing to the scattering of the light passing through the lens, or the polyopia which is produced by the formation of separate images by the different segments of the lens, vision is considerably improved under conditions which give rise to contraction of the pupil, so that some benefit is derived for a time by the use of a myotic or a stenopaic slit.

When a cataract is ripe, the vision is, as a rule, reduced to mere perception of light; but where the opacity of the lens is unaccompanied by any other affection of the eye, this perception is, except in the cases of extremely dense cataracts, very delicate. The simplest way of testing the light perception is to throw light reflected from a gas jet into the eye with the ophthalmoscope. The room should be darkened and the light be shaded from the patient. By turning the gas down or up it is easy to determine the smallest amount of light which is just distinguishable from the light of the room. Another way which is often practised, and which affords, too, a means of readily comparing the acuity of the light perception in different cases, is to hold a lighted candle in front of the patient in a dark room, and notice whether he distinguishes between light and darkness when the flame is shaded by the hand or allowed to remain uncovered. Except in the very densest cataracts, the difference should be readily appreciated when the flame is from fifteen to twenty feet distant from the patient's eye. Owing to the diffusion of the light which is caused by the opaque lens, the intensity of illumination is approximately inversely as the square of the distance at which the candle is held.

But as the diffusion is never complete, and a certain degree of refractive power always remains, the patient should also be able, where there is no complication, to appreciate fairly well the direction from which light falls into the eye. This may be tested by asking him to point out the position of the flame of a candle, held in succession at different parts of the field of vision. A good projection indicates almost always not only a tolerably free field of vision, but also, as the patient directs his fovea to the light, an intact central vision. The existence of very small central scotomata, however, may be overlooked, but such cases are extremely rare. When the light sense, tested with the gas jet and ophthalmoscope mirror, or the candle, appears defective, a more accurate test may be made by means of von Graefe's photometer. A very low value obtained in this manner is an almost certain proof of the existence of detached retina.

This loss of all vision, with the exception of light perception, which characterises most cataracts which are ripe for operation, must not always be taken as the criterion of maturity. In some cases of hard cataract there is considerably more vision than this; the patient, for instance, being often able to count fingers close to the eye, so that if one were to wait for complete blindness before operating, much time would be lost to the patient. Fortunately, although senile cataract almost invariably affects both eyes, there is often a considerable time intervening between the time when maturity of the cataract is reached in each eye, so that if an operation be undertaken as soon as the first cataract is ripe, the patient is not rendered absolutely helpless by the disease. When both eyes are pretty equally affected at a stage when most useful vision is lost, an attempt may be made to hasten the maturity of the one, or an extraction may be undertaken rather sooner than would otherwise be done. There are several ways of artificially ripening a senile cataract, none of which is very certain in its effect. One method—that which has been longest in use—is to make a small opening in the capsule with a cataract needle, at the same time slightly stirring up the cortex with the end of the needle. If this is done too freely it is apt to give rise to irritation, or even inflammation, which may necessitate an extraction under circumstances, too, which are not then so favourable for healing. It is frequently, more especially in the harder forms of cataract, of no avail. Another method consists in the performance of iridectomy, an operation which, when done with this object in view, or for other reasons when cataract is present, is called *preliminary iridectomy*. This is not only a harmless practice, but one which rather lessens the risk of the subsequent extraction. It is impossible, however, to foretell on which cases it will have the desired effect of hastening the completion of the opacity. Undoubtedly a greater number of cases are ripened when the iridectomy is combined with a kind of massage of the capsule, a proceeding recently introduced by Förster, and which may be performed either through the cornea, or, with proper precautions as to pressure and antiseptics, directly over the capsule itself. This is seldom followed by any irritation.

Sometimes the anterior capsule of the lens participates in the cataract, that is to say, the cells lining the inner surface of the capsule proliferate and lose their transparency, the mass formed having much the appearance of connective tissue with deposits of fibrin. A capsular cataract is more especially met with in lenses which have become shrivelled and over-ripe. It is rare that the opacity extends beyond the limits of a moderately sized pupil. If the cataract be examined, with the pupil dilated, before an operation is undertaken, the capsular opacity cannot well escape detection. It is intensely white, and frequently presents an irregular jagged outline. In cases of this nature the capsular forceps should be used, instead of the cystotome (see chapter on Operations), or, if the case be suitable, the lens removed in its capsule by Pagenstecher's method.

No certain, constant connection between senile cataract and any other disease has as yet been definitely established. There is an undoubted hereditary tendency, which appears to descend by the male rather than the female line, although the disease is no more frequent in the one sex than the other. Many individuals with senile cataract exhibit no other symptoms of senility; in others, atheromatous degenerations of the arteries are more or less evident. Diabetes sometimes co-exists with senile cataract. In most cases there is probably no connection between the two conditions, although there undoubtedly exists a true diabetic cataract. It has not yet been satisfactorily shown, however, how diabetes produces cataract. Only a very small number of diabetic cases are complicated by cataract, and repeated experiments make it appear pretty certain that the small amount of sugar contained in the nutrient fluids of the eye cannot be the direct cause. It is more probable, indeed, that the malnutrition and general cachexia give rise to the lenticular opacity in those predisposed to cataract, or in whom the conditions are otherwise favourable.

Etiology of Senile Cataract.—Some advance has been made in recent years in our knowledge of the etiology of cataract by the study of cataract produced artificially in animals. It has been found possible, by feeding animals on certain substances, to give them cataract. It is the observation in the living animal of the earlier stages of such cataract that seems to throw light on the causes of senile cataract. A very plausible explanation is thus afforded of how it is that in otherwise healthy eyes the lenses may become opaque and undergo the degenerative changes which characterise the condition of cataract.

So far as experiments have as yet gone, feeding with naphthaline appears to be the most suitable means of producing the artificial cataract. Naphthaline cataract was first described by Bouchard and Panas, but Magnus has the credit of having studied it in a manner which permits of inferences being fairly drawn with respect to the etiology of uncomplicated senile cataract. The first phase in the development of the opacity of the lens can be seen within ten hours of beginning feeding with 3 to 4 grammes of naphthaline per kilo. of animal weight. According to Magnus what is then seen is a number of very clear striæ passing from the equator of the lens towards its poles. These are found to be caused by little folds or depressions of varying depth on the lens surface. The folds involve the capsule. The lens fibres and epithelium are perfectly healthy and transparent. The folds are without doubt produced by shrinking of the lens. A similar appearance is brought about by drying the lens when removed from the eye in a hot chamber. This initial shrinking process is shown in Fig. 44. This first phase, in which the lens fibres retain their transparency, is shortly followed by a second,—the stage of opacity. An important fact in connection with the appearance of opacity is that it always begins in the same parts of the lens. It begins in two zones, one of which lies immediately behind, and the other immediately in front of its equator. Almost invariably the posterior zone is the one which shows the greatest degree of opacity, see Fig. 45. These zones of opacity gradually increase in breadth, spreading therefore from the equator towards the poles. In addition, however, there is a separate area of opacity to be seen at the posterior pole. As the opacity in this situation increases in size, it meets that which spreads from the posterior equatorial zone.

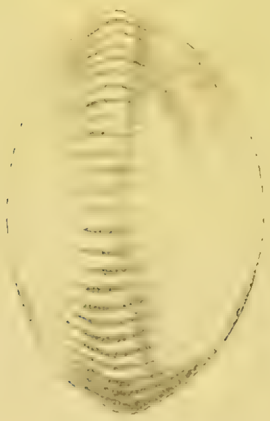


FIG. 44.—Appearance in lens produced by naphthaline, early stage.

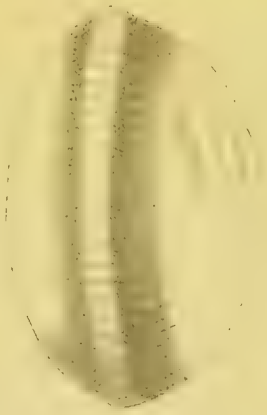


FIG. 45.—Appearance in lens produced by naphthaline, late stage.

If the naphthaline feeding be stopped in time, the opacities gradually and completely clear up. This takes place even if the whole posterior cortex is quite opaque. It takes place, too, always in the same way. The opacities begin to clear away simultaneously from the equator and the posterior pole. This process of clearing is due to the lens receiving its proper supply of normally-constituted nutrient fluid. That this is the case is evident from the fact that, if removed from the eye and placed in distilled water, lenses rendered opaque by naphthaline regain their transparency in a few hours.

The conclusion which may be drawn from the manner in which the opacity comes on and in which it clears off when the exhibition of naphthaline is stopped is, that the equatorial zones and the posterior pole are important regions connected with the flow of nutrient material to the lens.

Now, it has been pointed out by Magnus that about 93 per cent. of all senile cataracts, however much they may differ from it afterwards in their clinical aspects, begin in exactly the same manner as naphthaline cataract. But while naphthaline cataract is clearly the result of chemical changes in the nutrient fluid of the lens, such changes cannot well be assumed to be present in most cases of senile cataract. They undoubtedly play a part in some forms of cataract, more especially diabetic cataract.

Magnus suggests that in senile cataract there is first of all a greater or less stasis of the normal lymph in the lens, owing mainly to advancing cornification, but possibly also to vascular changes in the sources from which the nourishing fluid springs. This stasis may be supposed to affect more readily the transparency of the cortical lens substance the more its power of resistance is diminished by senile or other conditions. In this way he would account for the great differences met with clinically in the rapidity with which the opacity advances.

Broadly speaking, it seems not unlikely that cataract in man is due either to circulatory disturbances in the nutrient fluid of the lens, or, though less frequently, to alterations in the chemical constitution of the nutrient fluid.

The *diagnosis* of cataract presents no difficulties when no complications exist in the anterior portion of the eye, such as dense opacities of the cornea, occlusion of the pupil, &c. Before the invention of the ophthalmoscope, various objective and subjective tests had to be applied in many cases before the presence of cataract could be diagnosed. The most famous test was that known as Sanson's. This, which is based on the laws of reflection from curved surfaces, consisted in observing whether when a candle flame is held near the eye, and a little to one side of its optic axis, reflections could be seen from the two surfaces of the lens, as well as from the cornea. When the test is made with the normal eye, three images can be seen,—two erect, and following in the same direction any movements given to the candle flame, and one inverted, which moves in the opposite direction to the object. The nearest erect image is bright, and is caused by reflection from the surface of the cornea; the next image is also erect, though somewhat blurred—it is caused by reflection from the anterior surface of the lens. The third inverted image is reflected from the posterior concave surface of the lens, but it is so small and faint that it is not often easily seen without the aid of a convex lens. When one could satisfy one's-self that the second image was to be seen and yet the third was not formed, the conclusion was that the lens was present, but opaque. This test is seldom required now, but is sometimes used to determine whether the lens is present or not; if absent, there is of course only one reflection obtained, viz., that from the cornea. In very rare cases of extremely black cataract the diagnosis may be facilitated by resorting to Sanson's test.

By reflecting the light into the eye with the ophthalmoscope, we at once see, in the form of black patches or striæ on the yellowish-red background, formed by the light which reaches the observer's eye from the fundus, any opacity or opacities which may exist in the lens, while when the cataract is complete there is an absence altogether of any reflex from the fundus of the eye.

If the lens be merely examined by oblique illumination, and not dioptrically with the ophthalmoscope, the senile greyish reflection—in which, too, frequently the radii dividing the lens into sections are distinctly visible—is apt to suggest an opacity which does not in reality exist, and the transparency of the lens can be at once seen by observing the unbroken reflection of light from the back of the eye.

The only *treatment* by which sight can be restored without too great a risk of destruction of the eye is by extracting the opaque lens.

This is, as a rule, preferably done by shelling it out of its capsule, which must first be ruptured, after removal of a portion of the iris. If a piece of the iris be not excised, there is considerable difficulty in properly clearing the capsule of all the opaque cortical substances; the iris, too, is apt to be bruised during the expulsion of the cataract, especially if it be hard; and, what is of most importance, if an iridectomy be not performed, the iris is more apt during the early stages of the healing process to become entangled in the wound, and thus introduce difficulties which interfere with the perfection of the result aimed at.

The iridectomy may advantageously be performed five or six weeks, or longer, before the extraction, when circumstances permit. By doing so, there is undoubtedly slightly less risk of iritis following the operation. A preliminary iridectomy should always be done where there is a complication with glaucoma, and is also advisable in cases where the risks of auto-infection are greater than usual, as where there is dacryocystitis or chronic conjunctivitis. In certain cases the lens may be removed in its capsule. Operators differ as to the cases in which it is advisable to do this. There can be little doubt, however, that where any evident weakness in the ligament of the lens exists, and still more, where there is more or less marked subluxation, this is the best operation.

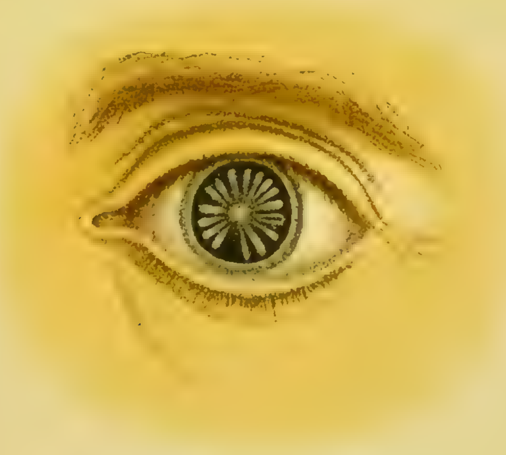


FIG. 46.—Case of incipient senile cataract, as seen by oblique illumination.

The methods of performing the different operations for the extraction of cataract are described in the chapter on Operations. The section which for many years has been most commonly adopted is one which lies as much as possible in the apparent sclero-corneal margin throughout. Since the adoption of proper antiseptic measures, so much stress is not laid on the exact size of section required, but there can be little doubt that, other things being equal, the larger it is the more likelihood is there for the dreaded suppuration of the external wound to occur.

After-cataracts form in cases in which the lens capsule has been left in the eye, and these opacities may attain such a density as to render a subsequent operation necessary. The secondary opacity generally forms after the lapse of some months, and is the result of a proliferation of the cells of the anterior capsule as well as an entanglement of cortical substance, which has not been removed at the time of extraction, or absorbed during the healing process. Where there is any inflammatory exudation this becomes matted, too, with the opaque capsule, and adds to the density of the screen thus formed. Undoubtedly, the density of the after-cataract depends to some extent on the manner in which the capsule has been ruptured at the time of operation, as well as on the completeness with which cortical lens matter has been removed. Much depends, too, on the rapidity of healing. In cases where the anterior chamber has been long in re-forming, there appears to be a greater tendency to the proliferation of the capsule cells, as well as to the retention between the anterior and posterior portions of the capsule of cortical matter, which is then not so readily absorbed.

The operations required for after-cataract are performed with the view to removing the opaque screen from the line of sight without removing it from the eye. When the main opacity is not in the axis of vision no operation is required. Where severe and prolonged iritis has followed the operation for the extraction of a cataract, there is not only a denser after-cataract formed, but the pupil is drawn up to the wound in the external coats of the eye, with the result that the vision is destroyed. If under these circumstances, however, there still remains some perception of light, it is possible, by performing the operation of iridotomy, to improve matters. The amount of vision which the patient recovers after such an operation depends greatly on the state of the vitreous. Often it is much clouded by exudation or the semi-organised remains of exudation, which after a time, if there is no fresh inflammation, may clear away to a very considerable extent. There is a great tendency to recurrence of inflammation, however, after iridotomy, which on this account should not be performed until the eye has come absolutely to rest. The opening should be made as large as possible, to prevent reclosing, should there be inflammatory reaction. This may easily enough be done when there is some retractile power in the iris, but in cases where the fibrinous deposit behind the iris is dense and tough, and the iris itself altered by inflammatory infiltration or atrophied, it is often extremely difficult. In such cases it is necessary to cut out a triangular-shaped bit of the opaque screen.

It is not generally advisable to undertake any after-cataract operation before the lapse of three months after extraction, and where there has been any marked inflammatory reaction after the first operation the eye should have been absolutely free from irritation for several weeks before anything further is attempted.

With regard to extraction, both eyes should never be operated on at the same time. This is a rule which should not be departed from, although there is often a temptation to operate on both when the operation itself has gone well in the first eye. The reasons are obvious. The operation is itself a more serious one when both are done; there is the possibility of some unforeseen accident happening, independent perhaps altogether of the nature of the operation, and avoidable on a future occasion, or some unfavourable condition, interfering with the success of the first operation, may necessitate a modification in the operation for extraction in the other eye.

Only a very few instances are recorded where a spontaneous cure of senile cataract has taken place, owing to absorption within the capsule. This is therefore so exceedingly rare, that it need not be taken into consideration in the treatment, and no medical treatment is known which will promote this natural cure. Occasionally a cure is effected by an accidental dislocation of the opaque lens.

After an extraction of cataract the eye does not immediately recover the full amount of sight of which it is capable, but goes on improving from day to day for a length of time, which varies considerably in different cases, usually, however, for at least a fortnight or three weeks. The cause of this improvement is to a great extent the absorption which takes place of the cortical matter left in the eye, and consequently it is slower, on the whole, the more incomplete has been the clearing away of the lens substance. In many cases, too, there are opacities in the cornea which only slowly clear away completely.

After the removal of the lens, the eye is of course left very much out of focus, and becomes highly hypermetropic. The condition of the eye when the lens is absent is called *aphakia*, and the degree of hypermetropia varies according to the build of the eye and the state of previous refraction. The higher the previous hypermetropia, the higher of course is the aphakic hypermetropia; whereas, on the other hand, there is less hypermetropia in an aphakic eye, the higher has been the previous degree of myopia. Sometimes the previous myopia has been exactly sufficient to neutralise the refractive change produced by the operation, so that emmetropia is the result, or there may even be some myopia left after the lens has been removed. Much the most frequently, however, convex glasses are required before any distinct retinal images are formed; and as with the absence of the lens there is an impossibility of any accommodation of the eye, absolutely clear images can, with the same glass, be received from objects at one distance alone. The practical result of this is, that at least two different strengths of glasses have to be worn, one for distant vision, and the other for reading. If the eye has been previously emmetropic, the glass which is required for distant vision is usually + 10·0, while for reading + 14·0 or + 15·0 suffice.

Vision is often improved by the addition of a cylindrical lens to the spherical one, as some astigmatism is frequently acquired, owing to a flattening of the cornea in a direction at right angles to the incision. It is some time before complete consolidation of the cicatrix leads to a final shape of the cornea, so that it is not advisable to prescribe the sphero-cylindrical spectacles until the astigmatism has acquired its permanent degree. There is some difference of opinion as to when spectacles may be worn. Reading should certainly not be allowed until three or four weeks have elapsed since all redness has disappeared from the eye. There is generally no harm in permitting the use of distant glasses a month after operation, if the healing process has been normal throughout.

Two forms of juvenile or *congenital* cataract are of common occurrence, viz.—*lamellar cataract*, which is a partial cataract, *i.e.*, one in which the opacity only involves part of the lens, and *complete congenital* cataract.

Lamellar cataract is probably most frequently congenital, although it undoubtedly often makes its appearance in early childhood. In the great majority of cases it exists in both eyes, though, as a rare occurrence, it is found in one alone.

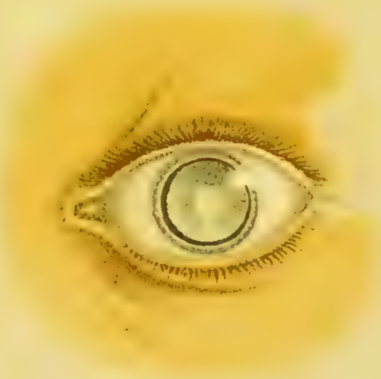


FIG. 47.—Case of lamellar cataract; oblique illumination.

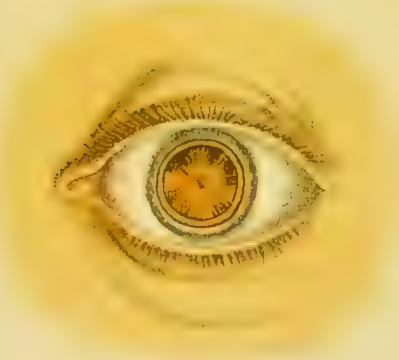


FIG. 48.—Lamellar cataract; ophthalmoscopic illumination.

The degree of visual disturbance which it causes varies very considerably according to the density of the opacity and the amount of lens substance which it involves. In some cases the density of the opacity is so slight that hardly any defect of vision is produced. In other cases, again, a high degree of blindness results from a dense opacity. Between these two extremes all degrees of visual disturbance are met with. The condition interferes to some extent, though probably never completely, with accommodation, and is often associated with generally only a slight degree of myopia.

When a lamellar cataract is examined by oblique illumination (see Fig. 47), the first appearance that strikes one is a tolerably uniform greyish opacity, which does not extend as far as the equator of the lens, and is more dense at its margins, from which often a few striæ pass towards the capsule. On closer examination the lamellar nature of the cataract can often be made out, that is to say, it is not difficult, as a rule, to satisfy one's-self that the opacity is limited to one or more contiguous lamellæ, within, as well as external to which, the lens is transparent. The cause of the greater darkness of the margin is the greater relative opacity which is caused by the anterior and posterior portions of the intransparent fibres meeting here. In this way, too, lamellar cataract differs greatly from other forms of cataract, which are usually darkest at the centre where the opacity is deepest. It is seldom that the opaque layers instead of being contiguous, enclose transparent lamellæ. The opacity occupies very different lamellæ in different cases, and consequently the diameter of the opaque area differs; there is never, however, an opacity of the layers immediately under the capsule. In some cases short striæ are to be seen, both in the anterior and posterior cortex. To these the Germans have given the name of "Reiterchen," owing to the way in which they, as it were, ride on the edge of the more diffuse opacity. They are, in fact, the equatorial portion of a commencing more peripheral lamellar opacity, which, however, does not always become complete. The cause of the opacity appears to be some arrest of development, at a time during intra-uterine life or early infancy, when the opaque layers were the most superficial. There is little doubt that it is a rachitic change, and is sometimes associated, as was first pointed out by Horner, with absence or defect of enamel on some of the teeth. I have most frequently seen this defect in the canines, and in one of the few cases in which I have observed the cataract confined to the one side, there was an absence of enamel on the canine on the same side, while in another case, that of the opposite side only was defective. In not a few cases of this form of cataract there is distinct evidence of heredity.

Lamellar cataract usually, though by no means invariably remains stationary. Cases exhibiting the equatorial striæ (Reiterchen) are more likely to progress to more complete opacity at some time or other than are those with a sharply defined edge. It is often overlooked until the patient attends school, and attention is drawn to it by the accompanying amblyopia or myopia. The amount of amblyopia is very variable in different cases.

The *operations* performed for this form of cataract are iridectomy, removal of the lens by discission, and extraction. Where there seems a likelihood of improving vision by displacing the pupil, which can be inferred when the vision, with proper optical correction if necessary, is improved by dilatation of the pupil, there can be no reason for abstaining from performing iridectomy, even when the amblyopia is comparatively slight. Removal of the lens, on the other hand, is attended not only with some risk—certainly very slight—but also introduces more inconvenience by rendering all accommodation impossible, and should not be undertaken unless vision is less than one-third of the normal. The choice of operation is in some measure influenced by the conditions as to probable progression of the opacity. Where there is good reason to believe that a recent deterioration in vision has taken place, which is not due to increasing myopia, discission may be performed even although the visual acuity is greater than one-third of the normal. In most cases for which iridectomy is performed, it should be done upwards, as it might be necessary at some later period to extract the lens. In this situation, besides, it is less unsightly, and less liable to cause any disagreeable dazzling.

Congenital total cataract is usually soft, and involves all the layers of the lens. It is frequently also complicated by a capsular opacity as well. These cataracts, too, undergo further changes in the course of time, and become shrivelled up and membranous, or in part calcareous. Degenerative changes, which take place in the suspensory ligament, often lead to their eventual dislocation. Alfred Graefe drew attention some years ago to a peculiar variety of congenital cataract, in which the opacity is mainly cortical, while a sclerosed nucleus occupies the centre of the lens. The best *treatment* for this form is to extract, just as in senile cataract. The more common form only requires discission, though, when calcareous patches exist, this has often to be followed very soon by evacuation of the swollen lens substance, as well as of the calcareous patches, through a small linear incision in the periphery of the cornea, owing to the irritation which the calcareous matter often produces when it lies in the anterior chamber. In the shrunken membranous cases, with opacity of the capsule as well, the most satisfactory result is obtained by dragging the whole out of the eye. This is apt to cause irritation unless done carefully. The membrane should be seized with a pair of capsule forceps, and very gently and slowly, and, as it were, coaxingly drawn out. If the resistance be very great, as much as will come without too great traction may be cut off with the iris scissors, and the rest left. Sometimes, instead of extracting, it is possible to get a good hole in the membrane with one or two cataract needles, or by cutting it across with a pair of de Wecker's scissors. The toughness of the structures generally, however, renders such efforts not very successful.

Secondary cataract most frequently begins with an opacity of the posterior portion of the lens—what is called *posterior polar cataract*—and is often very slow in advancing to other parts of the lens. The opacity is the result of malnutrition, and is most commonly met with where there is disease of the vitreous, retina, or choroid. An opacity beginning at the posterior pole should always raise a suspicion of being secondary, and it will generally be found that the vision does not correspond to the opacity, and that changes are to be seen with the ophthalmoscope in the deeper parts of the eye. An opacity of the posterior capsule rarely occurs, and is apparently mainly produced by deposition on it from the vitreous.

Stationary partial cataracts, in which the opacities are sharply defined and punctiform or irregularly linear in shape, and in different parts of the lens, are occasionally met with. They rarely produce any great defect of vision.

CAPSULAR CATARACT may exist without any accompanying opacity of the lens. It is most commonly in the form of what is called *pyramidal cataract,*—a conical protrusion of a central portion of the anterior capsule, which is at the same time densely opaque.

Sometimes pyramidal cataract is congenital, and then almost invariably bilateral. The pathology of this variety is not clear, but it seems to be the result of a retardation in the closure of the capsule. As a rule, however, the capsular opacity is set up when the lens is for some time pressed up against the back of the cornea, after a perforation due to ulceration. Under these conditions, a proliferation and fatty and fibrinous degeneration takes place in the cells lining the capsule, and the homogeneous capsule itself is protruded in front of this. In consequence of this origin, there is almost always to be seen some scar in the cornea indicating the position of the perforation. In many cases this is extremely faint, as the perforation has usually taken place in infancy, as the result of ophthalmia neonatorum, and the cicatricial tissue has gradually assumed an almost complete transparency. Often there is nystagmus in eyes with pyramidal cataract, and the vision is found to be more defective than can be accounted for by the limited opacity. The combination of pyramidal cataract with nystagmus is indeed almost a certain evidence of pre-existing ophthalmia neonatorum, or very early and extensive ulceration of the cornea.

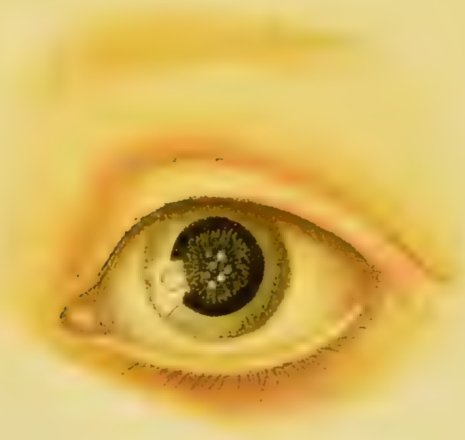


FIG. 49.—Punctiform cataract with capsular changes.

TRAUMATIC CATARACT.—The transparency of the lens depends, under normal conditions of nutrition, on the integrity of the capsule. As soon as there is a breach in its continuity, there is a great tendency for the lens fibres to become opaque. The most common cause, then, of traumatic cataract, is a wound of the lens capsule. A severe blow on the eye, without any penetrating wound, is—in rare instances, however—followed by cataract, which must consequently be considered traumatic. In such cases it is doubtful whether the capsule is ruptured or not. Probably in all cases where a rapid opacity takes place it is: indeed in some the rupture can be seen. In others, again, in which cataract slowly forms, the accompanying changes in the eye produced by the injury must be supposed to have in some way caused an interference with the nutrition of the lens.

It is extremely rare that the capsule is injured without being perforated, as it is not often that the body which has perforated the external coat of the eye penetrates just sufficiently to scratch the capsule and no more. Only two instances of an accident of this nature have come under my own observation. They both happened in the same manner. The eye was struck by a piece of iron, which ruptured the cornea and allowed the aqueous to escape, while small portions of rust remained in the corneal wound. While the anterior chamber was empty, and the lens lay up against the back of the cornea, the patients had rubbed their eyes, and the small portions of rust, which projected slightly from the back of the cornea, were thus scratched across the capsule, and remained attached to it.

If the hole made in the capsule be very small, it may sometimes heal without any opacity of the lens resulting. But even small ruptures, by admitting the aqueous humour into contact with the lens fibres, are apt to lead to cataract; an extensive rupture invariably does so, and some of the lens substance usually escapes into the anterior chamber, where it swells up, breaks down, and becomes absorbed.

The results obtained by treatment in the case of traumatic cataract are on the whole very much less favourable than for any other form of cataract. This is mainly on account of the liability that there is for other parts of the eye to be involved in the injury, or to become inflamed subsequently. Much depends, too, upon whether or not septic matter is introduced into the eye by the body with which it is wounded. The worst cases are those, of course, in which a foreign body is lodged in the eye. When the wound in the capsule is large, and a considerable proportion of the lens matter falls into the anterior chamber, a good deal of irritation is often set up, even when there is no septic element present. This is more likely to happen in adults, in whom the lens is harder and more irritating than in young individuals.

The *treatment* in all cases where the cataract has been caused by a perforating wound, should consist in bathing the eye frequently with a weak solution of corrosive sublimate, and in keeping the pupil well dilated, if possible, with atropine. Where there is much irritation, a careful examination should be made, in order, if possible, to determine the cause of it. If the rupture has been small, the lens is often found to be swollen in its capsule; and when this is the case, a larger opening should be made with a cataract needle, followed in a couple of days by removal through a linear incision of as much of the opaque lens as can be got away without any great difficulty. If the irritation be due to lens matter lying in the anterior chamber, a linear extraction may be performed at once. When it is evident that septic matter has been introduced, it is a good plan to combine the extraction with a good-sized iridectomy, and to freely irrigate the anterior chamber with corrosive sublimate solution (1 in 5000), afterwards using antiseptic dressings. An eye may sometimes be saved in this way, though at the risk of more or less permanent opacity of the cornea, owing to destruction of the epithelium lining its posterior surface. When complicated with prolapse of iris, the prolapsed portion should generally be excised, and the opaque lens matter as much as possible removed through the corneal wound. In cases where the irritation is slight, and tends to subside under simple treatment, it is, as a rule, better to leave the cataract alone until the eye has come completely to rest. Absorption often goes on without any interference, or may be hastened when slow, by stirring up the lens once or twice with the cataract needle. Even under apparently exactly similar conditions as to age, size, and position of capsular wound, &c., there are considerable individual differences in the time taken for complete absorption. This is the same, too, in the case of the diffuse traumatic opacity, which is produced by needling for lamellar cataract.

A traumatic cataract, instead of becoming absorbed, may undergo different changes, which are for the most part similar to those already described as occurring in other over-ripe cataracts. Very rarely there is the formation of true bone in the lens. In such cases it is always present in the choroid as well. Ossification never takes place unless the capsule has been ruptured, therefore only in traumatic cases.

Besides by absorption, there is another way in which traumatic cataract may undergo a spontaneous cure. A gradual clearing up of the opacity may take place. This is only met with, and that very rarely, when the capsular wound has been small and rapidly closed, while the opacity in the lens itself has been limited to the track of the body with which the eye has been wounded.

Traumatic cataract may be produced by wounds from almost every object which could possibly be imagined to be capable of penetrating the eye: knives, forks, scissors, needles, pens, &c., are the more common instruments which produce a wound without remaining in the eye. An extremely common manner by which the lens is wounded is with a fork, which is often used by children to unfasten their shoe-laces. This accident is most frequently a severe one, as, apart from the double wound produced by the two prongs, septic matter is generally carried into the eye.

It is a common enough thing for the patients themselves, or those who come with them, to deny any accident. When the presence of a cicatrix in the cornea renders it certain that there must have been one, and this is explained to them, they will then often admit it. A unilateral cataract in a young individual is, in fact, almost invariably traumatic, although some difficulty is experienced sometimes in finding any trace of the situation in which the eye has been penetrated.

Cataract produced by lightning has been observed, but as it is preceded by other disturbances of vision, it is more properly a complicated than a traumatic cataract in the clinical sense.

It is just possible, however, that the strong light may have something to do with its production. The lens absorbs the more actinic rays of light and may experience some abnormal molecular change which interferes with its nutrition when it is directly exposed to an exceptionally powerful light.

Attempts have been made to produce cataract in animals by subjecting them to strong light. Hitherto they have not afforded very conclusive evidence that the lens may be directly rendered opaque in this way.

Glass-Blowers' Cataract.—Glass-blowers are subject to cataract, which more frequently seems to affect the left eye, or, at all events, to begin in the left eye. I have seen several cases of this form of cataract. There can be no doubt that the occupation in some way favours the occurrence of opacity of the lens. Probably it is the action of powerful actinic rays. In one case which came under my treatment lately there was a large patch of eczema on the left cheek and left side of the nose, the cataract being also in the left eye. The curious preponderance of the cataract in the left eye is due evidently to the position in which the head is held in blowing. This position not only brings the left eye nearer the glowing glass, but brings the right eye under the protection of the nose.

DISLOCATION OF THE LENS.—In order that the lens may be displaced, some rupture or destruction or weakening must take place in some part of the zonule of Zinn. Dislocation occurs traumatically and idiopathically; in the latter case also congenitally. Dislocation, whether traumatic or idiopathic, may be complete or partial.

A partial dislocation or subluxation of the lens exists when some portion is tilted out of position whilst the centre remains in its place. The tilting may be of one side forwards, *i.e.*, round a vertical axis, or of the upper or lower edge forwards, or round a horizontal axis, or any intermediate axis of twist may occur. The partially dislocated lens often remains clear, and the diagnosis may consequently be difficult if the degree of displacement be slight. Astigmatism following a blow on the eye may be due to a slight displacement of the lens of this nature, and is often then associated at the same time with a slightly wrinkled condition of the capsule. When the subluxation is at all marked, a pushing forwards of the iris at one portion, and a consequent shallowing of the corresponding portion of the anterior chamber is observed, with a tremulousness of the iris in the opposite direction. There is then no difficulty in the diagnosis, especially when the pupil is well dilated, and the edge of the lens, often slightly serrated, is to be seen with the ophthalmoscope.

Complete dislocation takes place *into the vitreous*, or forwards *into the anterior chamber*, or, in case of rupture of the sclera, *subconjunctivally*. The first is the most common. The dislocation may be so complete that the lens is entirely removed from the line of sight, and lies at the bottom of the vitreous chamber, where it may remain for long transparent, though as a rule it very rapidly becomes opaque. When the vitreous is fluid, the dislocated lens bobs up and down with the movements of the eye. More commonly the dislocation is not so complete, so that the edge of the lens can be seen with the ophthalmoscope. A traumatic dislocation is very frequently associated with a separation of the peripheral attachment of a portion of the iris (irido-dialysis) or inversion of the iris, either complete or partial (see Fig. 50). Often in the case of idiopathic dislocation the lens has been previously opaque, and vision may even be improved by the dislocation. An idiopathic dislocation is mainly met with in cases of disease of the vitreous (see Fig. 51). The intimate connection between the zonule and the hyaline membrane of the vitreous brings about a participation of the ligament in disease of the vitreous. Dislocation into the anterior chamber is not nearly so common. It mostly takes place in cases where the lens is small and calcareous, but full-sized transparent lenses are occasionally dislocated in this direction. They often then remain transparent or semi-transparent for a long time. It would appear that a certain proportion of cases of dislocation of

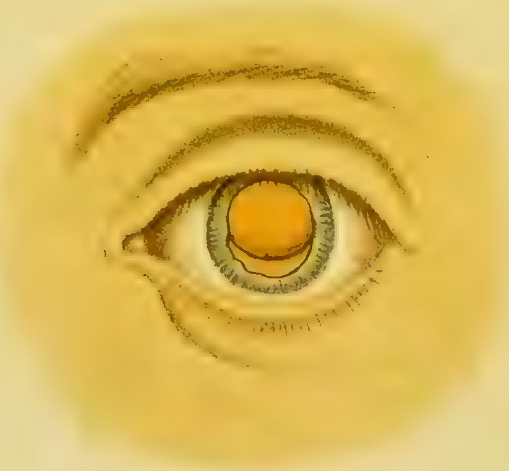


FIG. 50.—Traumatic dislocation of the lens from a blow at the point ×.

the clear lens forwards are cases in which there previously existed congenital displacement. In a number of cases of this accident which have come under my own observation, I have not, however, been able to confirm this connection. The two forms of dislocation described are very apt to set up irritation with increased intraocular tension. This is most likely to occur in the traumatic cases, and not, in my experience, more commonly in the one form than the other, although probably the eye of a young individual at least could more readily tolerate a dislocation backwards than forwards.

As to *treatment*, interference is only called for when the condition gives rise to irritation. The dislocated lens should then be extracted. In order to do this successfully care should be taken to make a sufficiently large incision. When the lens lies in the anterior chamber there is no occasion for attempting, what indeed I have always found impossible in the case of a full-sized clear lens, to avoid wounding it with the knife in making the incision, which should be downwards. Some vitreous generally escapes, too, but this cannot well be avoided. More difficulty is experienced sometimes in the extraction of a backwardly displaced lens. In such cases, after making a large incision, a scoop has usually to be used for removing the lens. Different methods of fixing the lens in some definite position before extracting have been devised, but these as a rule are not necessary.

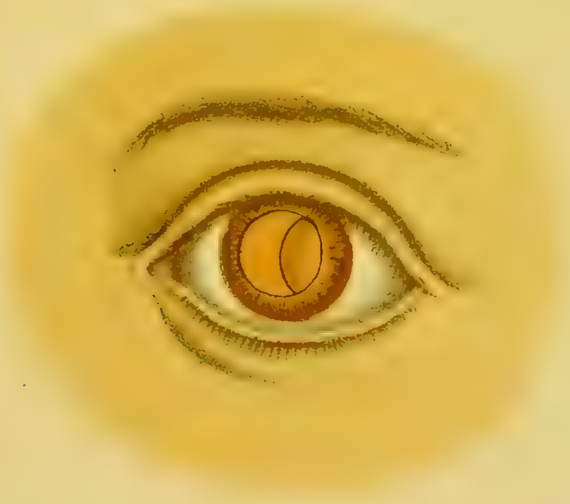


FIG. 51.—Idiopathic dislocation of the lens: ectopia lentis.

Subconjunctival dislocation (see Fig. 52) is only caused traumatically. It occurs almost invariably in adults, in whom the elasticity of the sclera is less than in young individuals. As a rule, if not invariably, the rupture of the sclera and escape of the lens take place upwards, and astonishingly little reaction follows the injury. There is generally a good deal of hæmorrhage at the time, so that the parts are not well seen, but the lens is easily made out, lying below the conjunctiva. When the wound in the sclera has healed, it has a dirty bluish colour, owing to an entanglement in the cicatrix of pigment from the ciliary body and iris.

The *treatment* consists in incising the conjunctiva, so as to permit of the escape of the lens. This should generally be done as soon as possible, especially if a portion of lens is included within the lips of the wound.

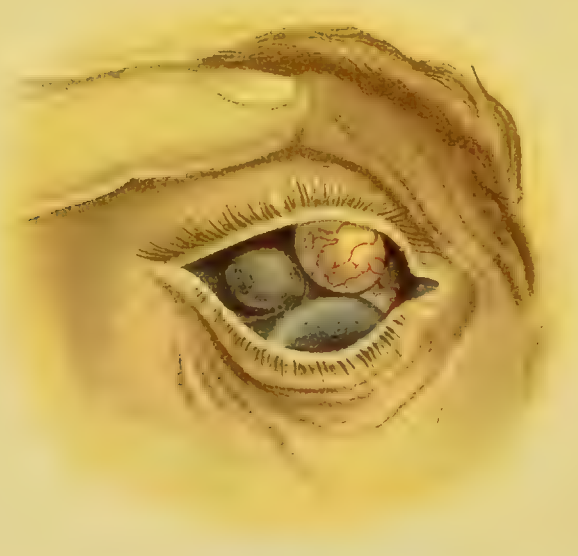


FIG. 52.—Subconjunctival dislocation of the lens.

Complete expulsion of the lens from the eye sometimes, though comparatively rarely, takes place as the result of accident. It is more common for it to escape through a large perforating ulcer of the cornea, after which the eye generally shrinks.

Congenital displacement of the lens, or *ectopia lentis*, occurs mostly upwards, or upwards and inwards, and upwards and outwards. It is often met with in several members of the same family, and is hereditary. The defects of vision depend to a great extent on the amount of displacement. As a rule, the patient directs his attention either to the images formed by refraction through the lens, or to those formed by the rays which reach the retina without passing through the lens. The manner in which he sees depends partly on the extent of the displacement, and partly on the state of refraction with or without the intervention of the lens. Usually the rays passing through the lens are too strongly refracted, those passing by the side of it too weakly refracted, so that in the first case there is myopia, and in the second hypermetropia. Whether the vision is most improved by concave or convex glasses can as a rule only be determined on trial. Sometimes they are equally good, but the one produces more diplopia than the other. On the whole, the monocular diplopia to which the condition gives rise is more evident when the refraction is corrected than when no glass is worn. The improvement in vision is, however, in some cases, very great, and the diplopia soon ceases to cause any annoyance.

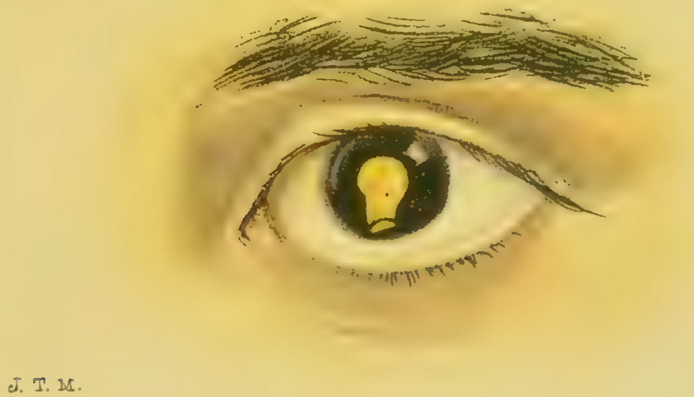


FIG. 53.—Coloboma of lens from a case of Sym's.

Ectopia is due to a faulty development of one portion of the zonule. It probably always occurs in both eyes, and is frequently associated with a defect in the power of convergence. A traumatic dislocation into the anterior chamber is more likely to occur where there is a congenital displacement than where the lens is in its normal situation. Notwithstanding the displacement the lenses remain transparent throughout life.

Two very much rarer malformations of the lenticular system are met with:—(1.) *Coloboma lentis*, or a defect in the lens. This only occurs in cases where a large and continuous coloboma exists of the choroid as well. And (2.) what has been called *Lenticonus*, and described as a conical protrusion of the lens, very much resembling keratoconus. In lenticonus we find, on illuminating the pupil with the ophthalmoscope, that every movement of the mirror causes crescentic lights and shadows to play round an enclosed circular area. Within this area, which is smaller than the pupil, retinoscopic shadows show myopia. Outside it there may be, and generally is found to be, hypermetropia.

Three parasites are said to have been found in the human lens—species of *Filaria, Monostoma,* and *Distoma.*

CHAPTER VI.

DISEASES OF THE RETINA AND OPTIC NERVE.

Diseases of the Retina.

Very considerable physiological differences exist in the degree of vascularity of the retina—that is, in the degree shown by the colour and appearance of the vessels. On this account, and also because the magnification of the ophthalmoscopic image is not sufficiently great for the detection of small differences in the size of the vessels, it is impossible to draw a hard and fast line between the normal state, and deviations in the direction of anæmia on the one hand, and hyperæmia on the other. For these reasons, too, as well as for others connected with peculiarities of the intraocular circulation, the observed condition of the retinal vessels does not afford by any means as delicate an indication, either of the state of the cerebral circulation, or of the heart, as might be, and indeed has been supposed.

Schultén, who has studied this question very thoroughly from the experimental side, has pretty conclusively shown that when there is increased afflux of blood to the brain without collateral hyperæmia, a similar afflux must take place to the ocular vessels. A passive hyperæmia of the brain, owing to impeded venous circulation, need not, however, affect the eye, as the ocular veins have other channels into which they can empty themselves besides the cerebral sinus, a thrombosis of which might therefore occur without influencing the circulation within the eye. When the venous impediment lies farther off, in the jugular vein or in the thorax, there will be at the same time a visible interruption in the circulation within the eye. Interruption in the carotid circulation affects the eyes, although the anæmia which this causes may not be apparent. In aortic stenosis, for instance, by maintaining pressure with the finger on the eye while examining with the ophthalmoscope, the prolonged wave of pulsation can be well seen and distinguished from the sharp beat which occurs under similar circumstances in normal conditions of the circulation. In aortic regurgitation there may or may not be spontaneous pulsation of the retinal artery and vein. This depends on the degree of compensation, or on whether or not there are other complications; if the mitral is also affected there is usually no pulsation.

Venous pulsation is often seen, most frequently on the disc, and is not necessarily indicative of any pathological condition. It usually, indeed, depends upon some interruption in the vessel itself, where it bends suddenly round an artery or some part of the nerve.

Spontaneous arterial pulsation is pathological, and indicative either of some general circulatory disturbance, sometimes only increased cardiac action, or of increased intraocular tension. When there is increased tension alone, what is seen is a quick flash of blood rapidly distending one or more of the more or less collapsed arterial trunks on the disc synchronously with each heart-beat. On the other hand, the pulsation sometimes, but by no means invariably, seen where there is some diseased condition of the aortic valve, is a comparatively gradual dilatation of the artery, producing often at the same time a visible movement of the vessel. This form of pulsation, too, is not confined to the trunks on the disc, but is often more distinctly seen at other parts, particularly where the vessels divide or make sharp turns.

Vascular changes in the brain need only appear simultaneously in the eye when the cause of such changes is a central one—that is, due to irritation or paralysis of the vasomotor centre in the medulla. An increase or diminution in the amount of cerebro-spinal fluid, giving rise to altered conditions of the brain, need not affect the circulation in the eye, though such conditions may be followed by other ophthalmoscopic changes. Pronounced ischæmia of the retina has been observed along with whooping cough; the condition is not permanent, and is probably spasmodic, although other possible causes have frequently been suggested to account for the marked diminution in calibre of the vessels.

Very definite changes in the colour of the blood have been met with in different diseases. In inflammatory conditions of the retina, as well as in all conditions of other organs where there is a rapid waste of tissue or defective oxygenation, the blood in the retinal veins may appear considerably darker than normal. The appearance is by no means always indicative of an actual darkening of the blood, but is often mostly, or it may be entirely, due to the greater contrast which is afforded by the swollen condition of the retina. In cases of severe diarrhœa, dysentery, and cholera, both arteries and veins have been observed to be much darker than normal. On the other hand, a lighter coloration of the blood is met with in chlorosis and other conditions which give rise to excessive anæmia or poverty of blood. In leukæmia it may be very pale or even greyish in colour, whilst in jaundice there is occasionally said to be a distinctly yellowish-green discoloration.

The streak of reflection along the arteries is brighter the lighter the colour of the blood. It is found to be narrow and less apparent when the blood is thick and albuminous, broader and more distinct when it is thin and watery. It is very doubtful, however, in how far any conclusion as to the state of the blood might be justified by the observation of this phenomenon, which appears to vary pretty much in distinctness under perfectly physiological conditions. Any marked œdematous condition of the retina, such as that which occurs from embolism of the central artery or commotio retinæ, causes the streak of reflection to disappear more or less completely. It is absent, too, in detachment of the retina.

Alterations in the size of the vessels occur in disease. Both arteries and veins may be either enlarged or diminished. It is most common, however, to find the calibre of the veins increased and that of the arteries diminished. Indeed, most inflammations of the retina cause this. Owing to localised phlebitis, some only of the retinal veins may be enlarged, and that often in an extreme degree. It is rare to find the arteries increased in size, whilst diminution in the size of the veins is only met with in the subsequent atrophic stages of retinal inflammation.

Sometimes, though very rarely, a formation of new vessels is seen to have taken place in the retina. Usually this, when it happens, is part of the process of organisation of an old blood clot. More frequently there may be seen leashes of small vessels passing into the vitreous and springing from the retinal vessels. A rare occurrence in connection with the vascular system of the retina is the anastomosis of some vessel either with another retinal vessel or with a choroidal vessel.

The retinal vessels seem to be very little liable to aneurism; only a very few cases, in which aneurismal dilatation has been met with to any extent, have been recorded. Perhaps the condition is not quite so rare as the paucity of observations during life might lead one to suppose; it has certainly been seen more frequently after death. The subject is, however, hardly of practical importance, though the fact of the rarity of retinal aneurism is not a little remarkable, considering that probably all forms of disease of the vascular walls occur in the vessels of the retina as well as elsewhere, and consequently, as will be seen, hæmorrhage from the vessels is far from uncommon. Probably the support given by the pressure of the fluid within the eye accounts for the general absence of such dilatations.

Hyperæmia of the Retina.—As has already been said, it is not always quite easy to say that the apparent congestion in any particular case exceeds the bounds of physiological variation. In coming to a conclusion on this point, it is well to pay attention to the relative sizes of arteries and veins. The calibre of a retinal artery is never, under altogether normal conditions, much less than three-quarters that of the corresponding vein.

Hyperæmia in the retina, as elsewhere, may be active or passive. An active hyperæmia may be set up by all such conditions as call for any unusual straining of the eyes, such as reading in the dark, or prolonged attempts at deciphering difficult manuscript or bad print. And this form of strain may be favoured, and more readily lead to congestion, when there is either some general weakness or some marked error of refraction which renders persistent exercise of this nature more irksome. The influence of the lower degrees of refractive errors in this respect has, however, been greatly exaggerated. Active hyperæmia of the retina, too, is met with along with conjunctivitis. Finally, it may merely be present as the first stage of an inflammation of the retina—*retinitis*—which either exists alone or is associated with inflammation of other of the deeper parts of the eye. As the normal relation in the size of the artery and vein is pretty well maintained in active hyperæmia, the diagnosis depends greatly on observing the state of congestion of the optic disc, which in such cases does not show so marked a contrast in colour from the surrounding fundus as is usual. At the same time, however, there is no indistinctness of its margins, such as occurs when inflammatory swelling of the papilla takes place. The cause of the deeper coloration of the disc is of course the springing more into view of the smaller vessels, and a similar excess in the number of visible vessels can also be made out by the practised observer in other parts of the fundus as well.

Passive hyperæmia, on the other hand, being due to some interference in the venous circulation, or in both venous and arterial circulation, gives rise to an abnormal relation in the size of arteries and veins. The veins are increased in size, sometimes to an excessive degree, while the arteries either retain their normal size or become diminished. The interruption to the circulation may be in the eye, as in the case of glaucoma, or external to it,—produced, for instance, by pressure on the optic nerve. Congested retinal vessels have not only their calibre increased, but are also lengthened. On this account they take a more tortuous course than normal, the tortuosity being greater at right angles to the retinal surface than in the surface of the retina itself. Contiguous portions of a vessel may therefore be at different levels, and consequently not in accurate focus at the same time. This undulatory shape causes the blood in some parts of a vein, when its direction is such that a greater column is looked through, to appear darker than at others where only a column the thickness of the vessel itself is seen, and this difference in the apparent depth of colour in different sections of the vessel is often enhanced by a simultaneous swelling and intransparency of the retina. Great tortuosity in the plane of the retina is most frequently only a congenital peculiarity. Passive hyperæmia is described by Leber as occurring in cases of congenital cyanosis, in which, too, the blood is of the same colour in both arteries and veins, and there is no interference with vision.

Anæmia of the Retina.—Some of the general conditions which are associated with anæmia of the retina have already been referred to. The changes are not sufficiently marked as a rule to afford any important evidence of the existence of anæmia of the brain, and indeed most cases of general anæmia are not associated with any marked ischæmia of the retina.

Anæmia, too, from excessive loss of blood, only produces temporary ischæmia of the retina. The ophthalmoscopic appearances met with in pronounced cases of ischæmia are great pallor of the disc and narrowing of the lumen of the vessels. There are besides altogether fewer retinal vessels visible over the fundus.

Ischæmia of the retina may be functional, when it is due to vasomotor constriction, or toxic, the effect of the poison being of the same nature, viz., causing constriction of the vessels by some irritation of the vasomotor centre. A most marked and not apparently very infrequent example of toxic ischæmia is that produced by overdoses of quinine.

There can be no doubt that it is the quinine and not the condition for which the quinine is administered that gives rise to the ischæmia. This was definitely proved by Barabascheff, who experimented on some of his colleagues. No doubt the appearances are due to poisoning of the vasomotor centres and consequent constriction of the peripheral vessels.

In the worst cases of quinine blindness, which are nevertheless recovered from, there is a persistence of the pallor of the disc and the narrowing of the retinal vessels. I have, for instance, seen a case in which the poisoning took place two years before the patient came under my observation, with such a complete absence of any colour in the disc, and such excessive constriction of the retinal vessels, that it recalled the appearances met with in many cases of complete optic atrophy. Nevertheless, in this case recovery had slowly taken place, and the visual acuity, colour vision, and light-difference perception had all but reached a normal standard.

The continuance of vascular constriction has been referred by Horner to endovasculitis set up by the prolonged spasmodic vascular constriction.

Much the more common cause of ischæmia is, however, the atrophic form, which occurs after optic neuritis, essential atrophy of the optic nerve, and inflammatory and other degenerative changes in the retina. Ischæmia is also more or less marked in cases of embolism of the central artery of the retina.

RETINAL HÆMORRHAGES.—Hæmorrhage from the retinal vessels is by no means an uncommon occurrence. The ophthalmoscopic appearances differ, as well as the degree of visual disturbances produced, according to the position and extent of such hæmorrhage.

Hæmorrhages are frequent in all cases where there is retinitis. Of other local conditions apt to be accompanied by retinal hæmorrhages may be mentioned:—Embolism of the central artery and thrombosis of the central vein, as well as probably sudden compression of the central vessels by hæmorrhage into the sheath of the optic nerve, glaucoma, atheromatous and hyaloid degeneration of the retinal vessels and traumata. But they are met with altogether independent of local inflammatory or other changes in many states where there is a diseased condition of the blood. The bleeding may be confined to the retina itself, or it may pass backwards and cause separation of the retina from the choroid, or forwards into the vitreous. This last result is not common, and only likely to take place when a large trunk is ruptured by accident or disease. Hæmorrhagic detachment of the retina is almost invariably traumatic. It occurs in cases where, with a tendency at any rate to hæmorrhage, owing to an altered state of the vessels, the intraocular tension is suddenly lowered by the escape of some of the

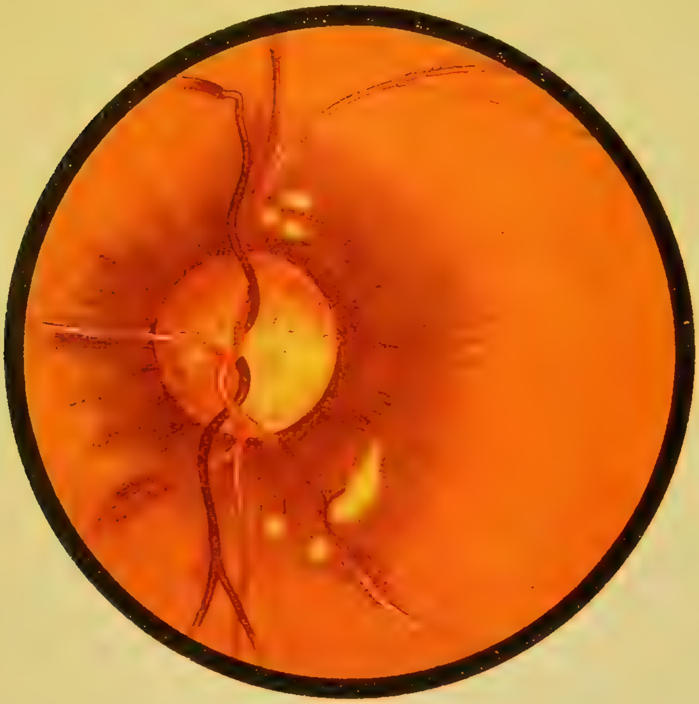


Fig. 54.—Retinal hæmorrhages from a blow on the eye.

contents of the globe. It occurs, for instance—fortunately only very rarely, however,—after cataract extraction. It is much more frequent after iridectomy for glaucoma. Hæmorrhages into the retina usually lie in close proximity to the vessels. When situated in the layer of the nerve fibres, which is very often the case, as it is in this layer that the larger vessels course, they have a tendency to be elongated in shape with sharply defined sides and irregular radiating ends. This form of retinal hæmorrhage is distinguished as a *flame-shaped hæmorrhage.* The peculiar shape is occasioned by the direction of the nerve fibres between which the blood settles and is constrained to pass. Only larger hæmorrhages in this part of the retina can assume a more irregular form, the extent of the extravasation being then sufficient to overcome the resistance of the nerve fibres which are pushed aside or ruptured. Deeper hæmorrhages in the retina are circular or irregular in shape, with a tendency rather to pass backwards, owing to the resistance offered by the connective tissue fibres of Müller, than to spread superficially. Hæmorrhages when numerous are usually found in the neighbourhood of the large vessels; mostly, therefore, near the disc. Frequently they are distributed in a radial manner round the disc. Less numerous hæmorrhages are not infrequently found in the region of the macula. Often, indeed, there is only a single, and, it may be, quite a small hæmorrhage in this situation. Occasionally a few hæmorrhages may be seen confined to some particular part of the fundus. It is not always easy to detect the reason for this, but sometimes it is due to localised phlebitis and thrombosis.

The subjective symptoms to which hæmorrhages into the retina, unassociated with retinitis, give rise, depend on their position and extent. When they are peripheral there are no evident symptoms, although if large there will be found to be a corresponding defect in the field of vision. When occurring at or near the macula they give rise to a positive scotoma, and sometimes, but comparatively rarely, to coloured vision. Thus, when fresh, patients may complain of a reddish cloud, which afterwards appears greenish, in front of their eyes. The objects seen in the region of the scotoma as it clears away may be distorted—that is, the patient may be conscious of more or less *metamorphopsia.*

Smaller retinal hæmorrhages are generally absorbed without leaving any trace and without undergoing any discoloration. Larger hæmorrhages generally leave some trace in the shape of degenerated patches, in which cholesterin occasionally forms, and gives rise to a very brilliant reflection. The appearances, therefore, of old hæmorrhagic spots as seen with the ophthalmoscope are very various. They may be mere patches of greater or less depigmentation revealing the choroidal structure behind. Or again, they may be yellowish-white or white, sometimes glistening white. These straw-coloured or white patches are often bordered by, and here and there covered by, masses of pigment. Patches of this kind are the remains of hæmorrhages into the deeper layers of the retina. It is often difficult to distinguish them from patches of disseminated choroiditis. Finally, there may be nothing but irregular pigment spots left, which are usually considerably smaller than the original extravasations.

Retinal hæmorrhages in old people, if due to an atheromatous condition of the vessels, may be followed by glaucoma. This is the so-called hæmorrhagic glaucoma, in which the prognosis is bad not only for vision, but also with respect to other apoplexies which may endanger life. According to von Graefe, if ten weeks have elapsed since the occurrence of the retinal hæmorrhages without any supervention of glaucomatous symptoms, these are not then so likely to come on. Small hæmorrhages often occur after iridectomy for glaucoma. These rapidly disappear, and are caused by the sudden relief of external pressure on the retinal arteries, which are probably always more or less diseased in glaucoma.

It does not appear to be known exactly how the smaller retinal hæmorrhages take place. Their proximity to the visible vessels makes it likely that they come from them, and not from the invisible capillaries. Yet there is rarely to be seen any evidence of actual rupture. On this account Leber has suggested that they are usually the result of diapedesis. Any tendency to rupture of the vessels, or to hæmorrhage altogether, is no doubt greatly counteracted by the intraocular tension, so that on this account any circumstance which sends the blood to the head is really not so dangerous in this respect as might at first be supposed. Active congestion is more likely to be associated with hæmorrhage than passive, hence the occurrence of hæmorrhage in menstrual disorders. But the most common immediate cause of retinal hæmorrhage is to be found in altered conditions of the vascular walls, which admit of rupture or diapedesis, or in altered conditions of the blood itself. Such changes are produced by inflammation, atheromatosis, scurvy, purpura, pernicious anæmia, leucocythæmia, nephritis, diabetes, and jaundice, in all of which conditions retinal hæmorrhages may be found. In cases where there has been an excessive loss of blood, a serous infiltration, giving rise to a white, somewhat opalescent, opacity of the retina, has been seen along with more or less hæmorrhage. From the appearance presented under such circumstances, it is not unlikely, as has been pointed out by Förster, that the cerebral symptoms which may occur at the same time may be due to much the same changes in the brain.

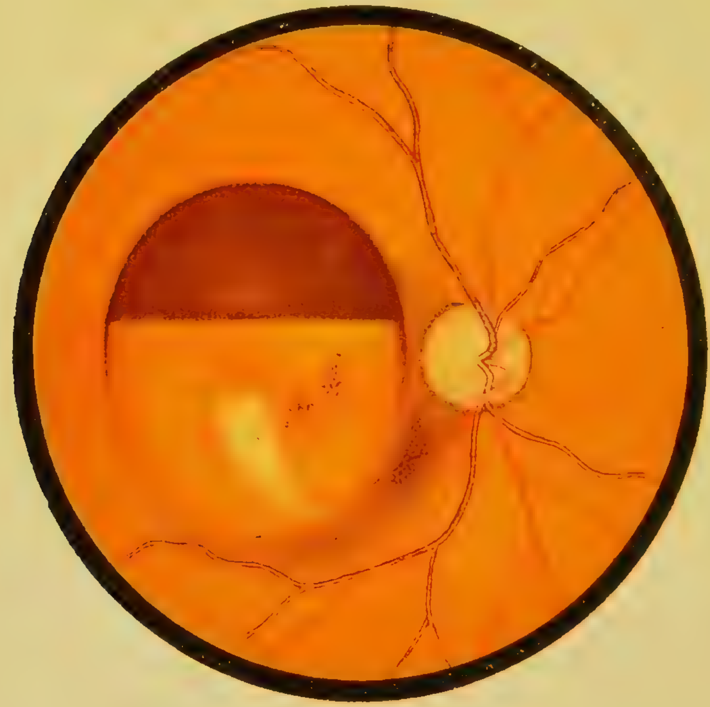


Fig. 55.—Sub-hyaloid hæmorrhage. Inverted image.

Sub-hyaloid hæmorrhage.—A somewhat rare occurrence, and one which gives rise to a very characteristic ophthalmoscopic appearance, is for an effusion of blood from the retina to spread out in a thin layer between the retina and the vitreous without passing into the latter. This all but invariably occurs at or near the centre, so that the macula is covered by a patch three or four times the diameter of the papilla. This patch of effused blood is at first dark red, with sharply-defined curved margins. After a short time it begins to undergo absorption, usually, if not always, from above, so that the line between the partially absorbed portion and that in which the blood is still present is perfectly straight and horizontal. A hæmorrhage of this nature causes a dense central scotoma, which passes off, either entirely or nearly so, after the lapse of several months. The prognosis, so far as the restoration of vision is concerned, is therefore good. The exact pathology of this affection, and more especially why the hæmorrhage should so often come in front of the centre of the retina, is not clear.

RETINITIS.—Inflammation of the retina occurs along with choroiditis and cyclitis, whether set up traumatically, sympathetically, or otherwise. Besides this inflammation, which is for the most part an extension from the other membranes, a primary retinitis is met with in many diseases in which there is an altered state of the blood or the blood-vessels. Of such may be mentioned septic processes and others which lead to thrombosis, syphilis, albuminuria, diabetes, and various forms of anæmia, possibly also oxaluria.

Prolonged exposure to strong light, excessive straining of the eyes in bad light, &c., in fact everything which leads to active congestion of the retina, while it no doubt favours the occurrence of inflammation under conditions in which it is likely to be met with, does not of itself suffice to set it up, and must be looked upon therefore as only of secondary importance in the etiology of retinitis.

Diffuse Retinitis.—A chronic diffuse form of inflammation of the retina, in which the ophthalmoscopic appearances are not very marked—merely hyperæmia of the disc, with a diffuse and sometimes more or less distinctly striated opacity of the surrounding retina—is that which is so constantly seen in cases of syphilitic choroiditis. Apart from the opacities of the vitreous, this appearance of the retina remains for a long time the only objective symptom of that disease. A very similar appearance in the retina is often met with, too, in cases of sympathetic ophthalmitis.

The subjective symptoms, prognosis, and treatment of diffuse retinitis depend in great measure on the accompanying changes in the choroid and vitreous, and are discussed under syphilitic choroiditis and sympathetic inflammation of the eye.

A curious and rare form of retinitis described by von Graefe under the name of central recurrent retinitis, appears also to be frequently of syphilitic origin. The ophthalmoscopic changes in this disease are often so little marked as to altogether escape detection; at other times a slight speckled opacity may be seen in the region of the macula. The affection mostly occurs in both eyes, but may be confined to one alone. The defect of central vision to which it gives rise occurs suddenly, and has a marked tendency to recur frequently, the intervals between each recurrence being characterised at first by complete, and afterwards by only partial recovery. The central scotoma has been observed by Nettleship and others to be ring-shaped at first. Much the same symptoms occur sometimes along with the more common syphilitic retinitis.

Purulent Retinitis is sometimes the starting-point of panophthalmitis, and especially, it would appear, in cases in which septic emboli form in the vessels. Clinically, however, there is nothing to distinguish this affection from purulent choroiditis, with which it is eventually associated whenever the suppurative process begins.

Hæmorrhagic Retinitis.—Many forms of retinitis are hæmorrhagic, that is to say, the changes in the retina are associated with hæmorrhages from the vessels. Only cases in which the number of hæmorrhages is very great and spread over the whole of the retina can be properly termed hæmorrhagic, as distinguished from other forms.

The term is rightly applied when the extravasations contribute a more important feature than the other changes, such as swelling of the papilla and retina, or the occurrence of patches of fatty degeneration in the retina, and where diseases of the kidneys and diabetes can be excluded. Retinitis, with abundant hæmorrhages occurring in one eye alone, is almost invariably of this nature. A greater or less extent of the retina may be covered by hæmorrhages, most of which are flame-shaped, and therefore superficial, though some are more irregular and deeper. Sometimes there is a hæmorrhagic extravasation into the vitreous, causing opacities in it. The retina itself has often a dull intransparent look, more especially around the disc. This appearance is produced by œdema. Patches of opacity, such as occur in other forms of retinitis, are sometimes seen, but they are insignificant in amount compared with the hæmorrhages. Their distribution, like that of the hæmorrhages, is radial. The disc, although its margins may be completely obliterated, is not swollen and prominent as in papillitis. More or less atrophy of the disc always comes on after some time has elapsed. The veins are greatly engorged and tortuous, the arteries usually narrowed and more or less in-

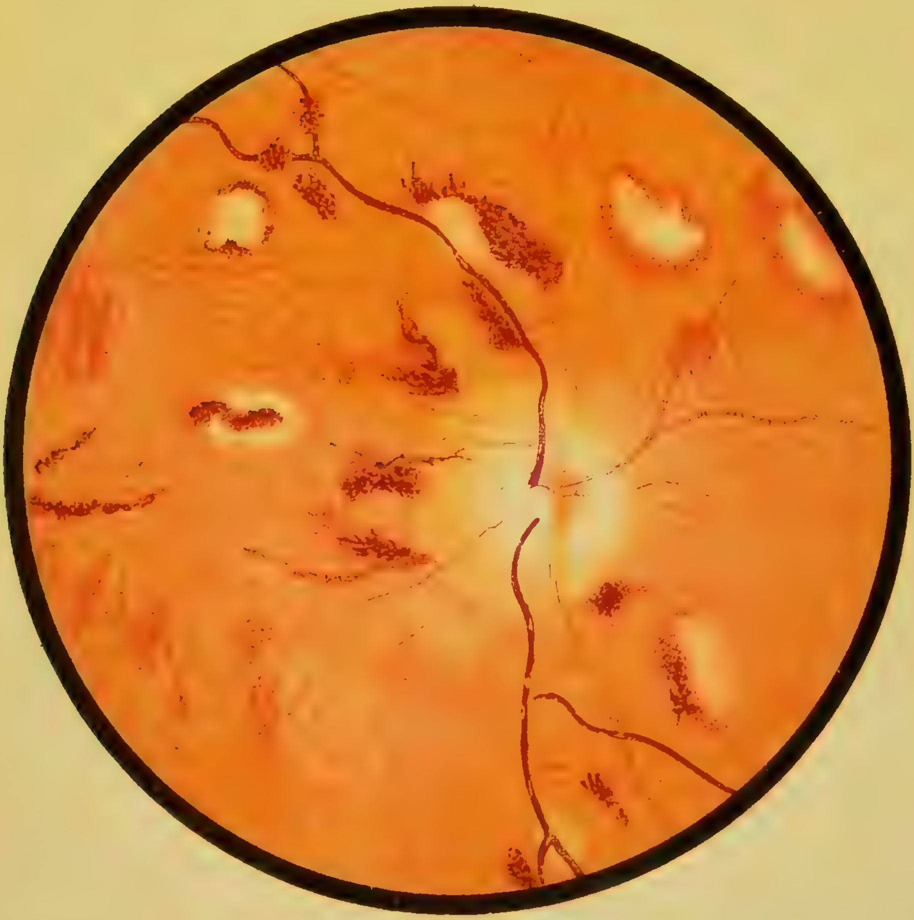


FIG. 56.—Hæmorrhagic retinitis.

visible. White streaks are often seen to accompany the veins. Occasionally the whole visible part of portions of the venous trunks are converted into white lines. This latter appearance is always met with where the hæmorrhages are confined to some particular sector of the retina. Sometimes there is a cardiac lesion, at other times not. I have never seen what I should be disposed to call true hæmorrhagic retinitis in both eyes—at all events, not at the same time. On this account it is difficult to believe that the hæmorrhages are due either to disease of the blood or of the vascular walls. Leber has suggested—although I am not aware that it has ever been demonstrated—that the affection may be due to embolic plugging of numerous smaller branches of the retinal artery. This explanation appears doubtful on account of the generally relatively normal condition of the peripheral vision. A more likely cause would appear to be venous thrombosis, the result of phlebitis associated possibly with an arthritic diathesis. While the central vision is as a rule very greatly reduced, the extent of the field of vision may not be at all restricted. After some time atrophic changes usually take place, so that although the hæmorrhages may clear away, leaving often little or no trace of their existence, the vessels become narrowed and obliterated, the disc pale, and the field of vision concentrically limited, just as in atrophy of the optic nerve, until eventually the vision is completely lost. The prognosis is certainly therefore extremely bad in the worst cases. Sometimes bleeding takes place into the vitreous as well, and this may be followed by painful glaucomatous symptoms.

No *treatment* is likely to be of much good. The eyes should be shaded from strong light, and reading or writing avoided as much as possible, attention being at the same time paid to the general health of the patient.

Albuminuric Retinitis.—A certain proportion of cases of Bright's disease, variously estimated by different authorities at from ten to twenty per cent., are associated with more or less blindness from inflammatory and degenerative changes in the retina. The so-called albuminuric retinitis met with in such cases is often so characteristic as of itself to render the diagnosis of kidney disease all but certain. At other times a retinal inflammation of undoubtedly albuminuric causation may not present symptoms which are at all characteristic.

The ophthalmoscopic appearances in albuminuric retinitis (see Fig. 57) are, speaking generally, more or less hyperæmia and swelling of the papilla, slight increase in the calibre of the veins and diminution in that of the arteries, and diffuse opacity of the retina, with hæmorrhages, and the formation of white patches, which are confined at first to the region of the posterior pole. The changes in the papilla are at first very slight, and there is some opacity of the retina, and a few hæmorrhages in the portion surrounding the papilla. The hæmorrhages, too, are mostly deep and round, less frequently flame-shaped. This appearance is of itself suggestive of the kidney being the cause, but it is not absolutely characteristic. At a later stage white, and for the most part deep-seated, patches of fattily degenerated exudation make their appearance in the retina, while at the same time there is often some swelling added to the hyperæmia of the papilla. These white patches are mostly found in the region of the macula, where they are arranged in a star-shaped form radiating out from the fovea. Around this central figure, the size of which is half again that of the optic disc or more, there are often a number of scattered spots of a similar nature. Such spots, too, are frequently to be seen in other parts of the fundus surrounding, and never at a very great distance from,

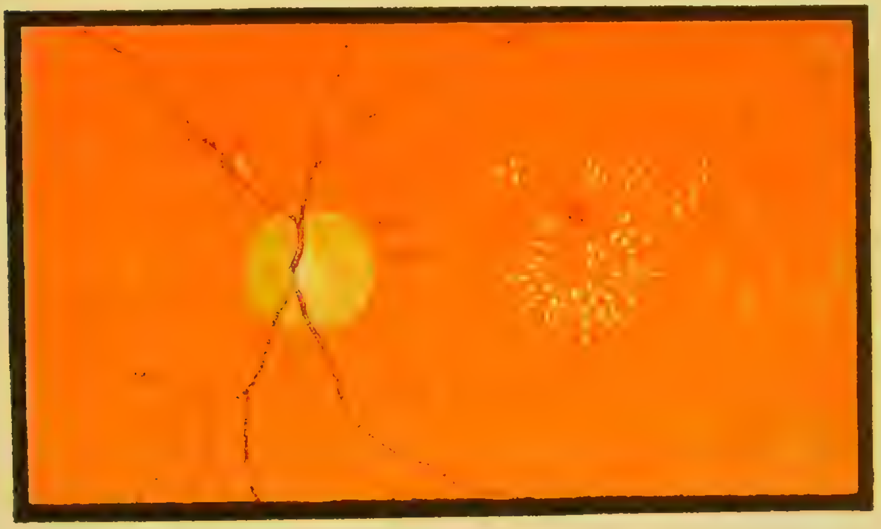


FIG. 57.—Albuminuric retinitis, early and typical stage.

the disc. In some cases the patches round the disc become confluent, and thus form an extensive white area or circumpapillary zone, stretching up to the disc on all sides, and with an irregular margin towards the more healthy retina. Extensive offshoots from this zone then generally accompany the larger vessels. The vessels in the circumpapillary exudation are seen to be large and tortuous, and are here and there altogether masked by it. The retina is thickened, and there are usually large and numerous hæmorrhages in the swollen white area. There is never any opacity of the vitreous. When the appearances just described are met with, it may be looked upon as altogether characteristic of albuminuria, because, although it seems certain that other causes occasionally give rise to identical changes in the retina, yet this happens so rarely, that it may be left out of consideration.

Little alteration may take place in the ophthalmoscopic picture, even after a considerable time has elapsed. The patches and hæmorrhages may slowly clear away, or fresh ones become deposited. At an advanced stage of the disease the spots are sometimes very thickly scattered over the fundus, and here and there, more especially at the centre, a dirty greyish diffuse pigmentation may develop in them. Occasionally, too, the white reflection from the patches becomes intensely brilliant and metallic looking, owing to the development in them of cholesterin. At the same time the papilla and the retina may be very greatly swollen, so that at places the vessels entirely disappear from view. The retinal swelling sometimes gives rise to the appearance of a detachment.

Most of the white patches lie in the deeper layers of the retina, the vessels of which may be seen to pass over them at places. Others are more superficial, and more or less completely obscure portions of the vessels. These appear to be caused by varicose swelling of the nerve fibres. Almost invariably both eyes are affected at or about the same time.

The degree of blindness produced by albuminuric retinitis varies very much, and depends on the extent of the pathological changes at the macula. There is therefore often considerable difference in the visual acuity of the two eyes. The disease rarely causes complete blindness, but most frequently produces so much amblyopia as to render reading impossible. The field of vision remains good, and also the colour vision. Sometimes the amount of visual defect does not correspond with, but is greater than can be accounted for by, the retinal changes. In such cases there may possibly be uræmic complications. Thus I have seen occasionally very marked deterioration occur without any apparent increase in the retinitis, followed after a few days by improvement, clearly pointing to some poisoning of the visual centres.

The *prognosis* in this disease is altogether gloomy, except in cases such as the albuminuria of pregnancy, where the condition may be transitory. Some cases improve so as eventually to recover useful vision. Most remain pretty stationary, and die not long, generally not more than two years, after the onset of the retinitis. An extremely grave symptom is apparent detachment of the retina.

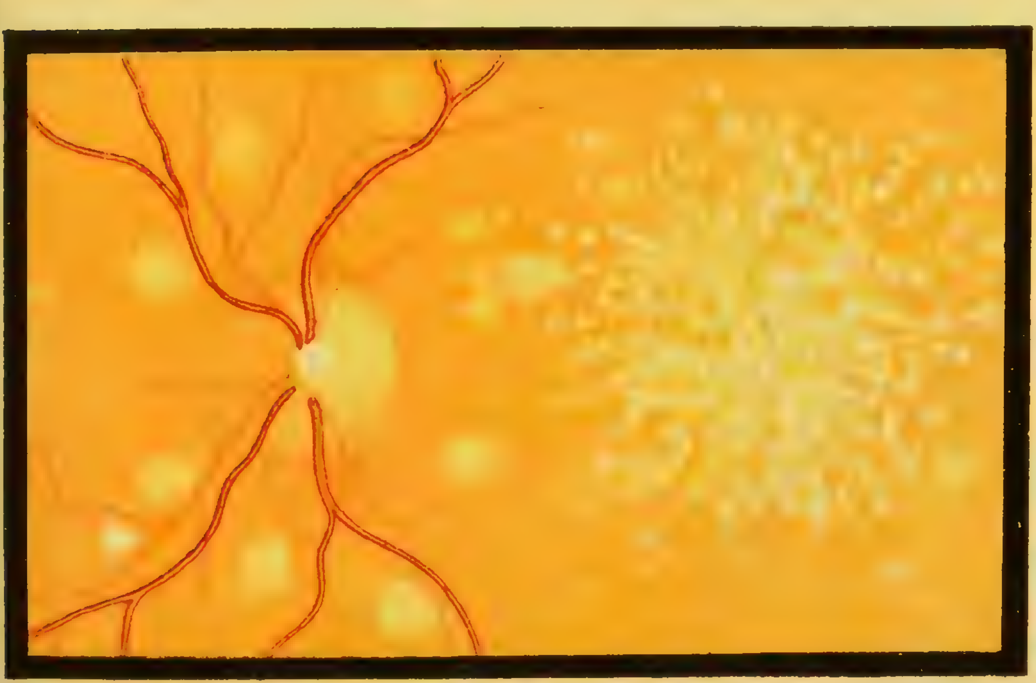


FIG. 58.—Albuminuric retinitis, late degenerative stage.

Albuminuric retinitis is mostly met with in chronic cases of kidney disease. As the general symptoms are then often little marked, it not infrequently happens that the diagnosis is first made with the ophthalmoscope. The patients are generally anæmic, and there is always albuminuria. It seems probable that the retinal affection is a direct consequence of the state of the blood, which arises from the presence of a diseased kidney. It is in fact a kind of chronic uræmia. It has not yet been shown what element of the blood is deleterious in this respect. The accompanying hypertrophy of the ventricle has been considered by some as the more direct cause of the retinitis. This view is untenable, because not only is there frequently hypertrophy without any retinitis, but many cases of characteristic retinitis have been observed without hypertrophy.

The *treatment* must be directed to the general disease which is the cause of the eye symptoms, the main indication being to relieve the kidneys by promoting the action of the bowels and skin. At the same time the usual precautions necessary in all deep-seated inflammations of the eye, viz., shading from strong light and the avoidance of reading, must be taken.

Retinitis in Diabetes.—In some cases of diabetes, mostly those of a severe type, there is a complication with retinitis. Diabetic retinitis is, however, very rare when compared with the albuminuric form. It is possible, too, that some cases described as diabetic have really been due more to accompanying albuminuria. Only when there is no albuminuria can the retinal changes be safely ascribed to other conditions of the blood more directly connected with diabetes. The form of inflammation is not characteristic, and very much resembles albuminuric retinitis, being, however, less severe, or at all events accompanied by less marked changes at the macula. The amblyopia produced varies very much from a slight defect of vision to complete blindness. The latter is due apparently only to complications: extensive hæmorrhages into the vitreous or glaucoma. Opacities in the vitreous are frequent. The *prognosis*, owing to the kind of case liable to be complicated by retinitis, is almost invariably bad.

Retinitis with circular spots.—I have seen two cases of a peculiar form of retinitis, in which white circular spots from $\frac{1}{8}$th to $\frac{1}{6}$th the diameter of the disc appeared in the upper half of the retina. The spots were not very numerous (in one case seven, in the other about twice as many). They were at some considerable distance from the disc, and distributed in an irregular manner. Dr. Ramage, who was at the time my clinical assistant, began to make a drawing of the appearances presented by one of these cases, but was unable to finish it, owing to the very rapid changes which took place. Dense vitreous opacities came on the next day. The same sequence of events were observed in the other case as well. Both cases were met with in adults with a syphilitic history, and as the beginning of a syphilitic choroido-retinitis. The circular spots in the retina may not unlikely have been collections of syphilitic matter. The area of the retina in which the spots were found was completely blind.

RETINITIS PIGMENTOSA. — A not very rare disease, the essential nature of which is, more correctly speaking, a sclerosis and pigmentary degeneration of the retina than an inflammation, has received the name of *retinitis pigmentosa*. The name is not altogether satisfactory, as the disease is not associated with pigmentary alterations until it has advanced to a certain stage, and the pigmentation may indeed occasionally never make its appearance at all.

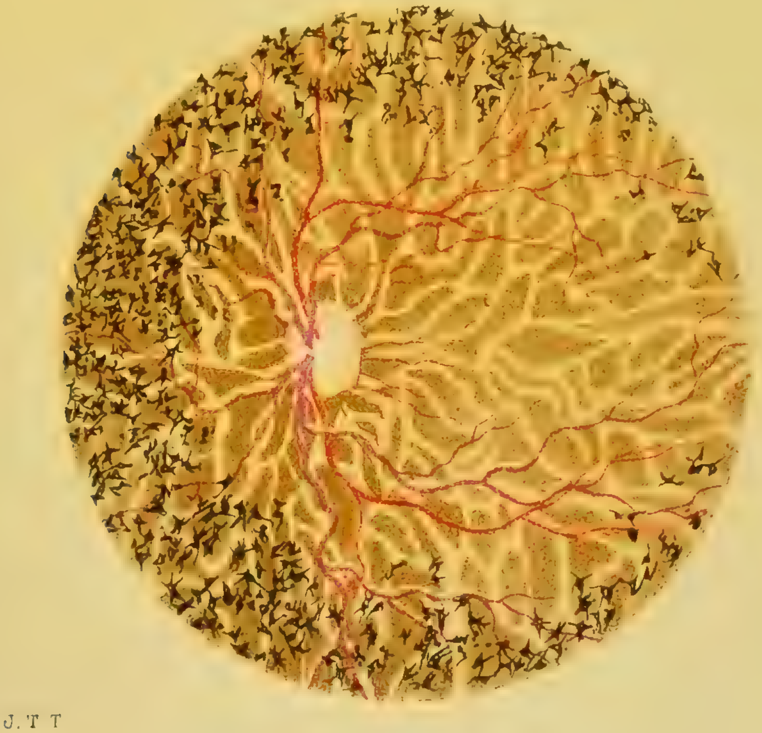


FIG. 59.—Retinitis pigmentosa.

Apart from the ophthalmoscopic changes, which are usually very characteristic, retinitis pigmentosa is accompanied by such distinctive subjective symptoms, that in the great majority of cases the diagnosis can be made without any objective examination. These symptoms are—night blindness, which has usually begun in early childhood, concentric limitation of the field of vision, with relatively good central vision, and a tendency to very slow deterioration.

The ophthalmoscopic changes are—a very equable, greyish pallor of the papilla, more or less marked narrowing of the vessels, both arteries and veins, usually a conspicuous defect in the pigment of the hexagonal cells, which allows the choroidal vessels, with their pigmented interspaces, to come into view, and a peculiar fine pigmentation in the retina. This pigmentation takes the form of intensely black bone-corpuscle-shaped patches, usually in close proximity to the vessels, and lying well forward in the retina.—(See Fig. 59). The pigmentation occurs mainly in a zone which lies midway between the centre and the periphery, and which is equally broad throughout. The breadth of the zone varies very much, being as a rule broader the more advanced and complete the sclerosis is. It generally approaches nearer the papilla on the inner than on the outer side, and in some cases patches may be seen quite up to the papilla. When very numerous, the thin filamentous processes of the pigment spots meet or come so close together as to form a network. The pigment comes from the layer of hexagonal cells, but is probably increased in amount by proliferation.

The manner in which the vision is affected by this disease varies somewhat in different cases. As a rule, while there is a pretty marked and sometimes excessive constriction of the field of vision, the central vision remains good enough for most purposes—for reading fine print, &c.—yet it is only rarely that there is full normal vision. Usually the smaller the field of vision retained, the worse is the central vision. It is only comparatively rarely that very small fields with good central vision are found, and in such the restriction may be observed to slowly increase without any marked difference in the central vision. Eventually blindness generally supervenes after the central vision has been destroyed, and a small eccentric portion of the field is alone left. In a good many cases in which there is marked concentric limitation, a portion of the temporal part of the field of vision towards the normal periphery may be found to have still retained some vision. Occasionally, too, a zone at the extreme periphery is left more or less unaffected, so that there is a broad band of the blindness, or a ring-shaped scotoma. A few cases have been described, too, in which there has been a central scotoma and good peripheral vision instead of the opposite condition. Two cases of this nature have come under my own observation, and in one of these the pigmentation was almost entirely confined to the region of the macula,

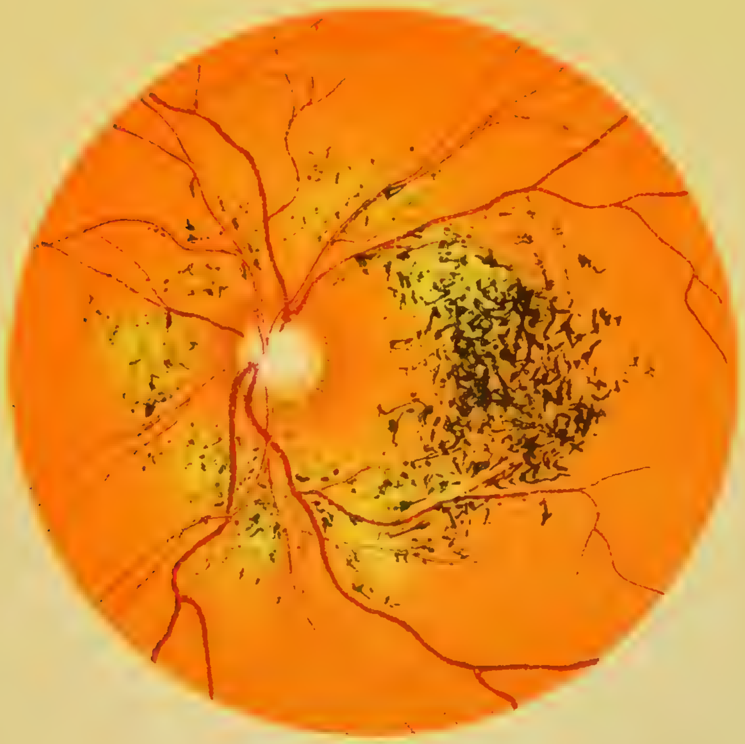


FIG. 60.—From a case of retinitis pigmentosa, in which the pigment is limited to the region of the macula.

where it formed a network in both eyes exactly similar to that usually seen in the more peripheral parts of the retina.—(See Fig. 60). But these, like the cases in which the pigment is altogether absent, belong to what may be called the atypical forms of the disease.

Altogether the pigmentation must be looked upon as having a subordinate significance in the changes which characterise this disease. It only makes its appearance after the disease has advanced to a certain stage, and may possibly sometimes remain absent throughout. The degree of blindness, too, cannot be inferred from the extent of pigmentation. On the other hand, the pigment is only met with in places where the sclerosis of the retinal connective tissue, which is the essential factor, has taken place. Yet there may be a sclerosis in parts of the retina where no abnormal pigmentation or depigmentation is visible. This is the case mostly in the central portions of the retina. The process of sclerosis may spread, and indeed most frequently does appear to spread slowly from periphery to centre, so that the absence of pigmentation at the centre is often merely owing to the less extensive alteration of the connective tissue there. In accordance with this the central vision is relatively greatly better than the peripheral. But the presence of very constricted vessels and of great amblyopia may be found with only peripheral pigmentation.

The pigment has been proved to come only from that contained in the retinal pigment cells, in which as one part of the degenerative process a depignentation took place. But there is no constant relation between this depigmentation and the amount of abnormal pigmentary deposition. This is very evident from the great differences seen with the ophthalmoscope (cf. *e.g.*, Figs. 59 and 60). The depigmentation is merely an atrophy of the retinal pigment, the abnormal deposition an attraction of the hyperplastic connective tissue for the pigment which, probably to some extent, proliferates (though this is not certain) in the new situation. The two processes are to a great extent independent, though primarily occasioned by the same cause — the sclerosis, by which, too, the nerve elements, but not, or to a less extent, the nerve fibres, are destroyed. It is not known what it is that gives rise to the differences in the extent to which both pigmentation and depigmentation are met with in any particular case. Both have a tendency to become more marked as time goes on.

The restriction of the field ordinarily met with causes some difficulty in orientation, as patients with this defect are in much the same position as any one looking through tubes held in front of either eye would be. Thus children frequently fall over things at their feet in a manner which to their parents appears, from their good vision otherwise, to be strange. The colour vision is good in retinitis pigmentosa, even in cases where the fields are very greatly restricted.

The light sense is affected both with respect to the appreciation of differences of intensity of illumination and to the vision in subdued light. The defect is most common and always most marked in the latter respect. It is this which causes the *night blindness*, which is a very early and very constant, though by no means invariable, symptom. Different degrees of the defect are met with. Often while the vision is so good in daylight as to cause no sort of discomfort, the patient is unable to guide himself in the dusk when the light is not so far reduced to make any very appreciable difference to the normal eye. This defect is often popularly called "twilight blindness." Things are seen by artificial light only when pretty strongly illuminated; thus while an individual might be able to read by the light of a candle, the surrounding objects in the room, which were only feebly illuminated by the light of the candle, might be more or less invisible to him.

In many cases the defect of vision produced by feeble illumination, though pronounced, is not nearly so bad as that just described, whilst in some it is hardly if at all noticeable. According to Leber, there are even cases where, instead of night blindness, there is a degree of hyperæsthesia of the retina accompanying retinitis pigmentosa which makes the vision in subdued light either more comfortable or actually better than in a strong, full light, a condition rather of day blindness than of night blindness.

The defect of the light sense is very probably due to changes in the hexagonal pigment cells of the retina. The other visual defects have been shown to be produced by alterations in the layer of rods and cones, the more particularly percipient elements. No doubt the nerve fibres are also affected, but the existence of ring-shaped defects in the field of vision points to the destruction of the rods and cones being the more important.

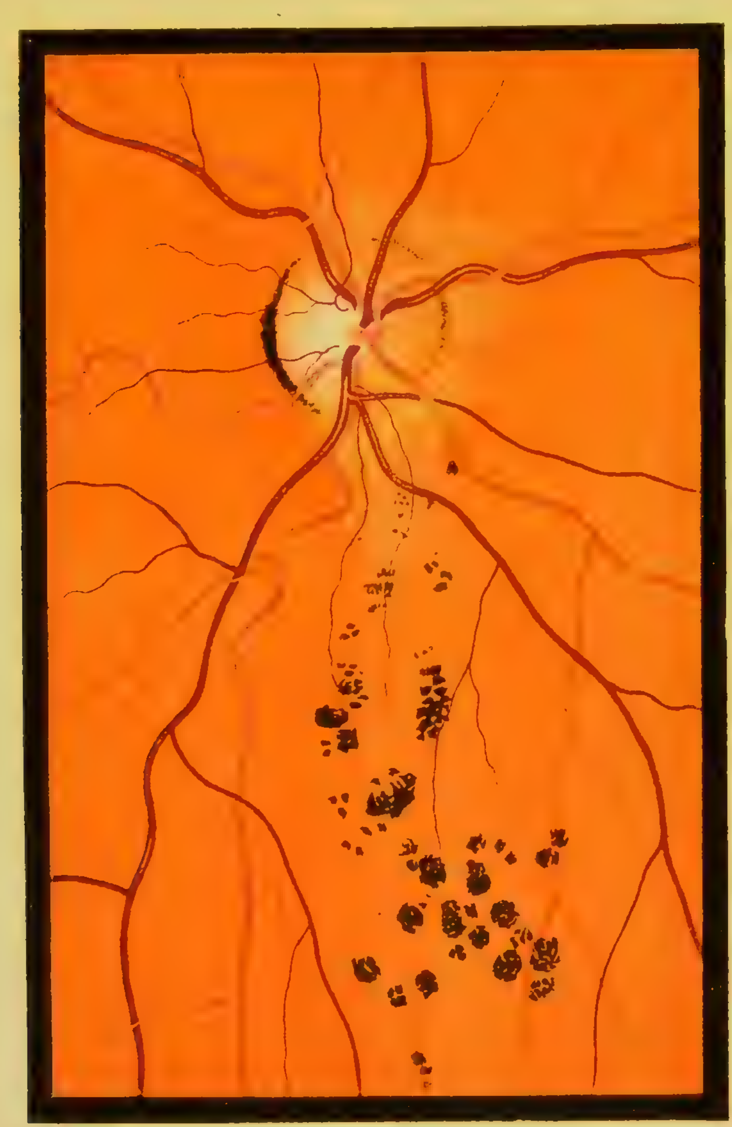
A form of stationary or very slowly progressive posterior polar cortical cataract is a pretty frequent accompaniment of retinitis pigmentosa. In the typical cases there appear to be no further complications; only where much the same retinal changes are met with, following inflammation from acquired or inherited syphilis, may there be more or less manifest alterations in the choroid, or iris as well. In such cases, too, the retinal pigmentation is rarely if ever so equally distributed as in the true disease.

There is sometimes a difficulty in distinguishing between cases of primary retinal sclerosis with pigmentation, and pigmentation of the retina which is secondary to choroiditis. Yet it is seldom there can be any doubt, as the choroiditis leaves deeper-lying patches which can be recognised as areas in which the choroidal tissues have been destroyed. In some long-standing cases of retinitis there is, no doubt, to be seen in the areas of depigmentation, what to all appearances is a sclerosis of the choroidal vessels as well. Again, I have met with a few cases in which there was undoubtedly both a true retinitis pigmentosa and a choroiditis, the latter being evidently an accidental and later complication.

Some cases of retinitis pigmentosa are atypical with respect to the picture presented by the pigmentation. The patches, instead of being more or less closely placed or interlaced, bone-corpuscle-shaped, and following the course of the blood-vessels and capillaries, are in the form of irregular, roundish, small masses distributed apparently without any definite arrangement in the deeper parts of the retina. The only difference in the pathology of those cases seems to be that the sclerosis and imbibition of pigment is more confined to the connective tissue framework of the retina, and less marked in the perivascular connective tissue than is mostly characteristic of the process. In other respects, and especially in the absence of any choroidal changes, there is no reason why such cases should be mistaken for later stages of choroidal inflammations.

Retinitis pigmentosa is almost invariably bilateral. I have only once seen a case in which it occurred in one eye only, while the vision of the other was in all respects normal. A few cases have been described by others. The spurious or syphilitic form is met with not so very infrequently in one eye alone, although it, too, is more frequent in both.

The disease begins either congenitally or in early childhood; occasionally not till some years after puberty. Some cases of congenital amaurosis, too, appear to be of the same nature. The pigmentation is probably never met with at birth, but usually makes its appearance during the first few years of life. The disease is considerably more frequent in the male than in the female sex. Thus Leber found the proportion about five to two (of 155 cases 111 were males, 44 females). It is unquestionably hereditary, and, just as in all other affections where this is the case, there is often consanguinity in the parents or grandparents. That consanguinity alone is not a factor of importance is evident from the fact that, as Macnamara has pointed out, the disease is frequent amongst the Hindus, among whom intermarriage is prohibited by their religion. Often several members of the same family are affected, rarely if ever all. Congenital deaf-mutism and idiocy are not seldom



A.W.H.

FIG. 61.—Congenital pigmentation of the retina (from a case of Mr. Sydney Stephenson's).

met with in the subjects of retinitis pigmentosa. Sometimes deafness comes on later.

The cause of the disease is altogether unknown. The fact that very similar appearances follow syphilitic inflammation is certainly suggestive of syphilis as a cause; yet it appears pretty certain that this is not the case. Most cases progress exceedingly slowly, and even remain for longer or shorter periods apparently absolutely stationary. No *treatment* can be said to be of the slightest avail.

A very rare form of disease, evidently closely allied to retinitis pigmentosa, has been called by Mooren *retinitis punctata albescens.* In this affection a number of small white dots are scattered over the retina, and there is night blindness. It seems probable that this may be an early intra-uterine form of sclerosis of the retina. Other atypical forms of retinitis pigmentosa occur. For instance, it is not so very rare to find no pigmentary changes of the characteristic form, and yet the appearance of the papilla, as well as the subjective symptoms, may be quite sufficient to justify the diagnosis "retinitis pigmentosa."

Congenital pigmentation of the retina.—A peculiar form of retinal pigmentation, which appears to be congenital, is sometimes met with. I have only seen two cases, but as it does not give rise to any symptoms, it may possibly be less uncommon than might be supposed. Fig. 61, which is from a case of Mr. Sydney Stephenson's, shows the characteristic distribution of the pigment, viz., in small irregularly-shaped groups occupying some sector of the retina.

Retinitis Proliferans.—This name has been given by Manz to a very unusual form of chronic inflammation of the retina. From the few cases I have seen, I think there can be little doubt that it is a well-defined clinical form of disease.

After a longer or shorter period of defective vision, the apparent cause of which is hæmorrhagic opacity of the vitreous, there may be seen, covering more or less completely the papilla and extending over the surrounding retina, a markedly prominent and deeply folded or furrowed bluish opacity. This fibrous or membranous looking opacity follows the course of the large vessels, which it to a great extent hides, but in the furrows may be seen vessels apparently newly formed. In the region about the main mass of connective tissue which this central opacity represents, may generally be seen whitish thread-like opacities stretching further towards the periphery of the retina. These are no doubt vessels surrounded by a dense opacity caused by perivasculitis. Hæmorrhages into the vitreous seem always to accompany this affection, and these, along with the cataract, which may subsequently make its appearance, make the image which it is possible to obtain with the ophthalmoscope, as a rule not very distinct. Possibly this hæmorrhage is in some way connected with the formation of the fibrous tissue bands. Cases of recurrent hæmorrhage into the vitreous are certainly afterwards found to present much the same appearances. As yet only one case, so far as I am aware, has been examined microscopically, and is fully described by Manz.

From his description of this case, there can be little doubt that the appearances were caused by a chronic form of inflammation of the retina, leading to an excessive hyperplasia of the inner surface of the membrane in the region of the main vessels.

I have only seen the disease in one eye, but it occurs apparently at least as frequently in both. The cause is unknown. In some cases there have been cardiac symptoms, which may possibly have had some influence. I have seen this, and also evidences of anterior sclerochoroiditis. A case is described by Mackenzie in the old *Ophthalmic Review*, which was probably one of retinitis proliferans, and in which there seemed to be a connection between the condition and oxaluria.

According to Manz, improvement may take place and be accompanied by partial disappearance of the ophthalmoscopic changes. He recommends *treatment* by mercurial inunction and leeching. Most cases are probably, however, little benefited by any treatment.

EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.—Not long after the important discovery of the embolic plugging of arteries by Virchow, a case of embolism of the central artery of the retina was diagnosed by von Graefe, and the diagnosis then made confirmed on post-mortem examination by Schweigger five years afterwards. Since that time the demonstration that the symptoms and ophthalmoscopic appearances usually referred to this lesion are really due to plugging, has been several times repeated, amongst others by Nettleship and Priestley Smith.

It is only rarely that the circulation is completely stopped in the retinal vessels; when this is the case, no pulsation can be obtained in the artery by pressure on the eye. The diminished supply of blood flowing into the eye causes the arteries to appear smaller, while at the same time the streak of reflected light along them becomes less apparent, or is altogether absent. The veins, too, may be smaller than normal, though this is usually the case only with those portions which are on and near the disc. They are often rather distended further towards the periphery and taper towards the disc. The ischæmia is rendered further apparent, too, by the invisibility of the smaller vessels. What looks like, and is often described as, spontaneous pulsation, may be seen in the arteries, due to the partial interruption of the blood flowing into them, producing a more or less marked difference in the degree of their distension with every systole. The intermittent passages of broken columns of blood may sometimes be observed in the veins when the circulation is not altogether, but all but, stopped.

Besides the varying appearances met with in the vessels indicative of circulatory impediments, an embolism of the central artery produces other and more characteristic changes in the retina, which, however, last only a short time after the accident. The central portion presents a whitish opaque appearance, not unlike the bloom on fruit. This appearance is due to œdema. It is most intense round the macula and optic nerve, and often indeed hardly appreciable elsewhere. As a rule, too, in the area occupied by the œdema it does not present an equal density or opacity throughout, but the whiteness tends to fade off at about equal distances from the papilla and macula. Thus, midway between the macula and the disc, it is seldom if ever so dense as in the vicinity of either. The œdema has, too, a more streaky appearance round the disc. At the centre of the macula, and corresponding in position to the fovea centralis, there is no opacity. Owing to this, the appearance presented by this portion of the retina is very remarkable, and generally described as a *cherry red spot.* So strong is the contrast between the colour of the fovea and the surrounding retina, that this appearance cannot fail to at once attract attention. The cherry red spot in the middle of the œdematous opaque retina, when taken along with the history of sudden

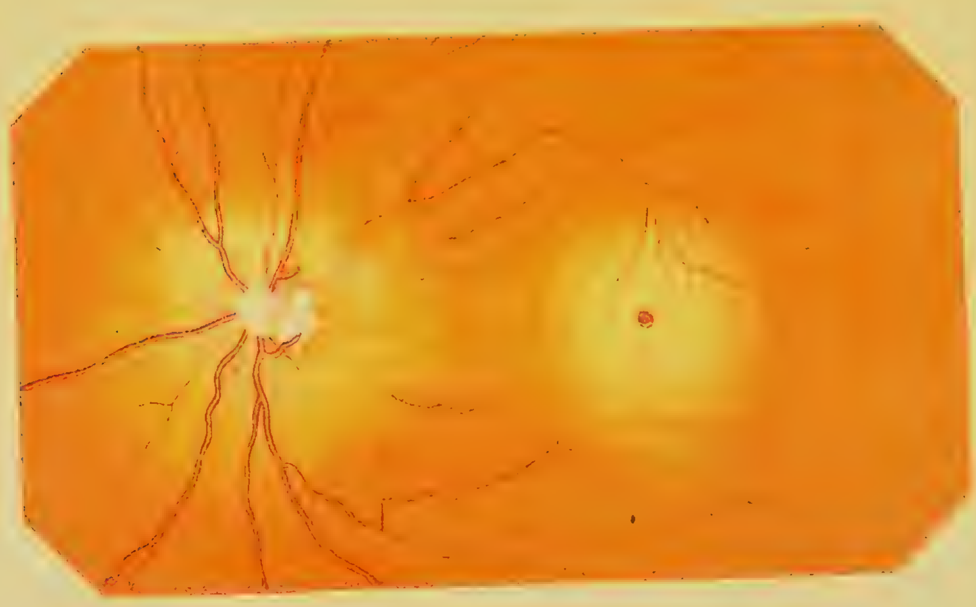


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FIG. 62.—Embolism of central artery.

and spontaneous loss of sight, is perfectly characteristic of an interruption in the circulation. A similar change, though rarely if ever so definitely marked, may follow severe blows on the eye.

Whether the interruption in the circulation in any given case is due to embolism of the artery, or to thrombosis in the vein, or hæmorrhage into the sheath of the nerve, causing compression of the nerve and vessels, is not always easy to determine. The subjective symptoms are the same in all causes of interruption. The first cause is probably considerably the most common. There are apparently two forms of thrombosis, the phlebitic and marastic. Angelucci, who had an opportunity of verifying in four different cases the diagnosis of thrombosis made during life, gives the following points as of importance in the differential diagnosis. *Embolism of the central artery:*—Normal course of vessels, arteries narrowed, veins gradually increasing in calibre towards periphery, no venous pulsation, absence or scantiness of retinal hæmorrhages. *Thrombosis of central vein:*—Tortuosity of vessels, arteries of normal calibre or nearly so, veins gorged with blood and here and there interrupted, venous pulsation, retinal hæmorrhages. Certainly some cases of embolism are accompanied by hæmorrhages, but they are never very numerous, and we should expect in thrombosis to find this feature more pronounced.

The œdema of the retina does not come on immediately on the plugging of the vessels, and takes several hours, or even a day or two, to reach its height. It then slowly passes off, leaving no trace after a week or two. The diagnosis may not then be so easy unless the appearances in the vessels are marked. It will depend more on the history and the nature of the visual defect.

Embolism of the central artery causes sudden, and in most cases all but complete, and permanent blindness. So sudden is the loss of vision, that patients are only conscious of having all at once become blind in one eye. If it occurs during the night, they find on waking that they have lost the sight of one eye; if during the day, they may be conscious of a rapid veiling of the sight, which has become quickly denser and denser, until after the lapse of not more than a minute, all sight has gone. Occasionally the eventual blindness is preceded by a kind of aura—an obscuration—followed by complete recovery. This may even take place several times at longer or shorter intervals before the blood supply is definitely and finally cut off. In a few cases some amount of vision is regained, but it rarely if ever happens that the patient recovers anything like useful sight.

The suddenness of the blindness produced by the cutting off of the blood supply to the retina is a remarkable illustration of the close dependence of its functional activity on its blood supply. The absence of anastomosis with other trunks, too, is the cause of the blindness remaining permanent, so that only where the plugging is incomplete from the first can any subsequent improvement be looked for. No visible necrosis of the retina takes place. This is a somewhat remarkable fact, but is probably accounted for by the blood supply to a great part of the retina being drawn from the choroidal vessels.

In a number of cases a small portion of the temporal side of the field of vision retains some vision. Usually this is only slight, merely amounting to appreciation of the movements of the hand in this position. This circumstance is of some importance in the diagnosis in a difficult case when all distinctive objective signs have disappeared. When taken along with the history of sudden onset, and the presence of some cardiac lesion, it would render the diagnosis of embolism not unlikely.

Occasionally the embolism, instead of settling in the main trunk, may plug one of the branches of the central artery. There is then produced a defect in the field of vision corresponding to the portion of retina supplied by the plugged vessel. This is a much rarer accident than embolism of the trunk. I have seen one case, and had an opportunity of observing the appearance produced a few hours after the infarction took place. The position of the embolism could be distinctly seen by the sudden narrowing of the artery on the other side of it. The surrounding retina over an area equal in size to the disc was œdematous, and presented the same appearance as the macular region in the blocking of the central trunk. A well-marked scotoma existed in the field, corresponding closely to this œdematous area. This disappeared in the course of a few days, and there was no appreciable permanent interference with vision.

Possibly some such cases are not really cases of embolism, but of localised spasmodic contraction of retinal arteries. The existence of a scotoma corresponding to the œdematous area is a significant fact which has not yet received sufficient attention.

In most cases there is some affection of the heart, but it is not always possible to diagnose this with certainty. Both eyes are never blinded by embolism at the same time, and cases in which the accident happens first in one and then in the other are of extreme rarity.

While the *prognosis* is therefore absolutely bad as regards the affected eye, the other may be looked upon as perfectly safe. There is more danger of embolism occurring elsewhere, and leading to other paralysis.

Very little can be done in the way of *treatment* for embolism in the retinal arteries. Kneading or massage of the eye is recommended, and is said to have dislodged the clot and led to improvement in a few cases. There can certainly be no harm in trying this. I have done so several times, but without success. Iridectomy and paracentesis of the aqueous chamber are also recommended by some, with the object of rapidly reducing the intraocular tension.

DETACHMENT OF THE RETINA.—Cases in which, either as the result of accident or of disease, the retina becomes detached from the choroid, are of pretty frequent occurrence. The separation takes place between the hexagonal pigment layer and the layer of rods and cones, the former remaining attached to the choroid.

Detachment of the retina, though its existence was known to surgeons in the pre-ophthalmoscopic times, was rarely diagnosed,—only, indeed, when the detached portion lay so far forward that it could be seen through a clear crystalline lens. The disease was considered uncommon, and was described by different authors, amongst others by Wardrop, who figured a case, as "dropsy of the choroid coat." Very soon after the invention of the ophthalmoscope cases were diagnosed by the aid of that instrument, and it was not long before von Graefe gave a very complete clinical description of the disease.

Detachment of the retina may be primary, that is to say, unaccompanied by any apparent inflammation, or secondary to affections such as cyclitis, which gradually lead to shrinking of the eye. Tumours springing from the choroid, too, give rise to detachment. In some rare cases the detachment occurs where there is inflammation of the retina itself, as in albuminuric retinitis. Such cases are always described at all events as cases of detachment, but it is by no means certain that the retina is actually detached. Probably in most, if not all, the appearance is due to excessive infiltration of the retina itself, so that its inner surface comes to occupy a position which causes it to appear detached. In traumatic cases the separation may take place at once, or not until some time has elapsed. Idiopathic detachment of the retina comes on either suddenly or gradually; the first is more common.

The objective appearances met with are usually sufficiently characteristic to render the diagnosis with the aid of the ophthalmoscope easy. Occasionally, owing to transparency of the detached portion, or to this circumstance combined with shallowness and small extent of the detachment, the nature of the disease can only be made out with certainty, if at all, by very careful examination. Often, too, a difficulty in the ophthalmoscopic diagnosis may arise from the presence of opacities in the lens or vitreous. The diagnosis is made by observing that some portion of the fundus is seen to disappear suddenly out of focus, and often at the same time to be altered in colour, sometimes to a bluish, at others to a greenish grey, according to the manner in which the light is reflected from its surface. When the vessels on the surface of this detached portion are focussed for, they appear darker by direct examination, and often smaller than normal. The detached retina, too, may as a rule be seen to shake or even sway about with the slighest movement of the eye. When the retina remains clear, owing to the absence of any turbidity of the fluid behind it, as is often the case in the beginning, and sometimes, especially in young individuals, for long after it is detached, no indication may be afforded by alteration of colour. The diagnosis will then depend upon one's finding an area which, in order to be distinctly seen, requires an alteration in the focus. On examination by the direct method, the interposition of a weaker concave or a stronger convex glass will be necessary; while by the indirect method the observer will have to withdraw his head and the interposed convex lens more or less. At the same time the vessels over this more hypermetropic area are blacker than in the immediate neighbourhood where the retina is in position. A parallactic movement of the vessels over the red fundus can often be made out too, more especially if a choroidal vessel can be seen behind the detachment. It is rare that the detachment is so shallow all over that the diagnosis cannot possibly be made with certainty by paying attention to these points. On the other hand, the retina is sometimes pushed so far forward that it can be seen through the media of the eye without the ophthalmoscope.

The dark appearance which the vessels on the detached portion of the retina so often present is due to their being seen to a great extent by transmitted light, just as the striæ in an incipient cataract appear black when examined with the ophthalmoscope. The diminution in the apparent size of the vessels when seen by direct ophthalmoscopic examination is accounted for by their lying in front of the focal plane of the eye. It is consequently more marked the deeper the detachment.

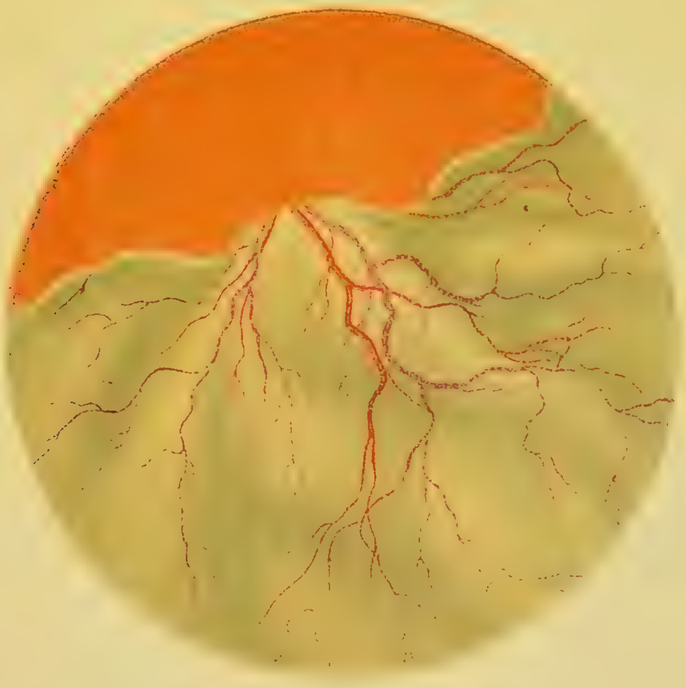

Fig. 63.—Detachment of retina.

At some portion of the detached retina there may often be seen a rent or rupture in it. This varies very much in size and has a very characteristic appearance, allowing the choroid to be seen with perfect distinctness through the opening. The margins of the rent may sometimes be seen turned in towards the vitreous. The occurrence and appearance of these rents, which were first observed by von Graefe, have recently been shown to have important bearings on the pathology of detachment of the retina.

In some cases, especially where the detachment has been hæmorrhagic, there may be seen irregularly disposed straw-coloured striæ or star-shaped opacities in the retina. These might be taken for scars, did they not frequently appear to interlace and lie behind perfectly intact vessels. They are in all probability fibrinous deposits at the back of the membrane.

Von Graefe supposed at first that although a sudden serous exudation might possibly take place, the fluid producing detachment was as a rule blood. This view was shown to be erroneous, when opportunities occurred for anatomical examination. It was then discovered that most frequently the subretinal fluid is serous, of a clear straw colour, and exceedingly rich in albumen. It sometimes becomes turbid, and may contain cholesterin. Thus Nettleship has described a case where crystals of this substance were found in the anterior chamber, having passed through an aperture communicating with the subretinal space.

Atrophic changes are found to take place in the retina which is detached. It may at the time of examination have lost all its nerve elements and be converted into a mere connective tissue membrane. Very often, on examining an eye in which there has been an old detachment which has become total, the retina is found to be separated in the shape of a funnel or convolvulus with its apex at the optic nerve and its base at the lens and ciliary processes.

In almost all cases where the detachment has existed for some time, and is not produced by tumour, the intraocular tension is found to be diminished. This may or may not be the case at the time the retina becomes detached. In a few cases, even although there is no tumour present, the tension of the eye is increased instead of diminished. This may be due to complication with iritis and total synechia, or there may be no visible inflammatory changes in the anterior part of the eye, but a true glaucoma. I have several times seen glaucomatous excavation of the papilla co-exist with detachment of the retina. This may possibly be merely a coincidence of the two affections, between which there may be no relation.

The *subjective symptoms* vary according to the portion of the retina detached, the state of vision in the other eye, and the rapidity with which the detachment is developed. They are—defective central and peripheral vision, metamorphopsia, and night blindness. Often for a longer or shorter period, before any actual displacement has taken place, the patient complains of muscæ or black spots of different shapes and sizes floating in front of his eyes, and of subjective light sensations, flashes, and rays of light, coloured as well as uncoloured. When the detachment does occur he is often conscious of a cloud coming in front of his field of vision. This is more likely to be observed in cases which come on suddenly and in which the vision of the other eye is lost. It is almost always noticed where the detachment is caused by tumour, as in such cases the central vision is, as a rule, relatively much better than in those in which the disease is idiopathic. At the same time the outlines of objects appear distorted. The apparent irregularity, too, which they present is apt to be inconstant, and to change much like the images reflected from the wavy surface of disturbed water. The distorted objects often appear to be bordered by colour.

There is usually more or less night blindness produced, the light sense being greatly reduced, both with respect to the minimum amount of light visible, and the perception of differences in illumination. A remarkable tendency to confuse between blue and green is often met with. This may possibly be due, as Leber suggests, to the absorption of the blue rays by the yellowish subretinal fluid; or it may be caused by absorption by fluid lying in front of the retina.

In cases of detached retina, where the condition has lasted for some time, there is usually a marked contraction in the field of vision corresponding to the portion of retina detached. As the recently detached retina retains its function to some extent, the restriction of the field in fresh cases may often escape observation unless the examination be made in subdued light. The boundaries for peripheral colour vision are narrower than the line separating the undetached from the detached portion of the retina, that is to say, the field of vision is more restricted for colours than for white light. The defect in the field of vision has most frequently a more or less indefinite, ill-defined, and irregular boundary. If the field be at first restricted below, which is frequently the case, an extension upwards of the restriction is not unlikely to follow, corresponding to the tendency to gravitation of the subretinal fluid. In this extension, either or both sides of the field in the neighbourhood of the point of fixation may be involved, even the fixation point itself.

In almost all cases of simple detachment of the retina the central visual acuity is more or less diminished. This is due no doubt to an œdematous condition at the macula. When the detachment is caused by tumour the defect in the field is usually much more strongly defined, whilst the central vision may remain unaffected. This point may be of some diagnostic importance in a doubtful case.

When detachment of the retina occurs idiopathically, it is frequently found in both eyes. Only occasionally, however, does the detachment occur simultaneously, or nearly so, in the two eyes. Generally a considerable time, often many years, elapses before the second is affected. It would be of considerable practical importance if one were able to tell in what cases the retina of the second eye was likely to become detached, and what appearances and symptoms, positive or negative, would indicate a safe prognosis in this respect. Probably when the diagnosis of detachment of the vitreous—which there seems every reason to believe always precedes the retinal separation—becomes more easy, an advance may be made in this direction. The condition of the light sense may possibly afford information, too, on this point. The occurrence of subjective light and colour sensations, and a notable increase in the number of muscæ, is always a suspicious circumstance.

Detachment of the retina is more frequent in old than in young individuals, the number of cases increasing with each decade up to sixty at all events, as has been shown by statistics collected by A. Pagenstecher, Poncet, and others. It is for some reason or other more frequently met with in men than in women. It occurs in eyes presenting all the different states of refraction. Taking the average of a number of German statistics on this point, I find that in rather more than sixty per cent. there is myopia, or in about three out of every five cases. Horner's statistics showed fifty-seven per cent., and in this country, where myopia is not so common as in Germany, the preponderance of myopic cases, if it exists at all, is certainly not so marked. With reference to the frequency of the disease in myopia, statistics by Hortsmann and Schleich show it to occur about once in every thirty cases in Berlin. This is certainly rather high, as cases of simple myopia are often not seen by an oculist at all, and besides, it would indicate a greater frequency of the disease than is actually found in most places.

The retina may be detached at any part, but in a large proportion of cases some part of the upper half is detached. Thus, of one hundred and twenty-six cases occurring at Göttingen, only thirty-nine were found not to involve some portion of the upper half of the retina. In the course of time the lower portions as well are apt to become detached by the gravitating of the subretinal fluid.

Cases which have existed for some time are very often complicated with cataract, and the opaque lens not infrequently undergoes subsequent calcareous degeneration. Opacities in the vitreous are sometimes met with too, and this is almost always the case where the detachment has been caused by hæmorrhage, either spontaneous or following injury to the eye. In these cases the opacities are often membranous.

The *prognosis* in detachment of the retina is always bad. Most cases go on to complete blindness, but as a rule, in idiopathic cases, the eye retains its shape and does not give trouble. Sometimes inflammatory changes make their appearance and end in shrinking of the eye; at other times the subjective sensations give rise to much discomfort. In a very few cases improvement takes place owing to the retina becoming reapplied, but even after this is permanent the vision is not fully restored. All that one could possibly expect, indeed, would be for the detached portion to adhere sufficiently firmly to prevent the hitherto undetached part becoming separated, and allow of the improvement taking place in it which might be possible from the disappearance of any œdema. A certain amount of inflammation would almost be necessary to effect this. Occasionally there seems to be a marked tendency to repeated recurrence of a clear detachment. The pathology of such cases may possibly be different from the usual form.

The *pathology* of detachment of the retina is not yet quite understood, although the researches of Leber and Nordenson have recently gone far to clear up the matter. Much more is, however, required before the explanation given by them can be regarded as applicable to all cases. The difficulty has always been to explain how a sudden effusion of fluid could take place below the retina without giving rise to increased tension, or why indeed, as the intraocular pressure must tend to keep the retina pressed against the choroid, there should be any possibility of its becoming separated, unless some sudden loss of tension by perforation of the globe took place. Two main views have been held for a long time. In the first place, the detachment was ascribed to a process of shrinking in the vitreous. This had early been recognised as the result of anatomical examination by Arlt and Müller in cases where the detachment was secondary to cyclitis. A similar cause was also apparent where a separation took place some time after hæmorrhage into the vitreous, as here the contraction of the blood clot might drag on and detach the retina. But it was not easy to understand how, with a normal or nearly normal transparency of the vitreous, there could be exerted by it a traction which could give rise to a spontaneous detachment of the retina, and yet this sudden detachment in an eye in which the media were clear was the most common case calling for explanation. The second explanation lay in assuming that an exudation took place from the choroid, and by accumulating between it and the retina caused the separation of the retina. This view was apparently supported when Raehlmann propounded the hypothesis that such an exudation might readily be supposed to take place if the detachment were preceded by some alteration in the chemical constitution of the vitreous, which gave rise to an interference in the process of diffusion taking place between the blood in the chorio-capillaris and the presumably saline solution occupying the vitreous chamber. The untenability of this hypothesis, however, is apparent, as Nordenson and others have pointed out, from the fact that the vitreous chamber contains exactly the same albuminous fluid as lies behind the retina. Besides, it ascribes to the living retina *in situ* the same properties, as far as osmosis goes, as a dead membrane, on which assumption it would be difficult to understand why it ever retains its position. Stellwag seems to have been the first to maintain that there must be some diseased condition of the vitreous to admit of the possibility of detachment of the retina, while the trembling or shaking which can usually be seen in the detachment would only be possible if there were a fluid both before and behind it. A definite step in advance was made by Iwanoff, who showed that the retinal detachment was always preceded by a detachment of the vitreous, the subvitrinal space being occupied by fluid. After this discovery Wecker came with the hypothesis that the subvitrinal fluid found its way behind the retina as soon as a rent occurred in that membrane.

Leber, who had found that a process of fibrillar shrinking took place in the anterior portion of the vitreous leading to its detachment, and that the margins of the rent or rents found in the retina were always turned in towards the vitreous, came to the conclusion that the shrinking vitreous, while maintaining its transparency, dragged on the retina, eventually causing a rupture. When this rupture took place he supposed that the fluid in the vitreous then passed in behind the retina, through the rupture, and filled up the space left by the membrane, which was drawn forwards at the place of its attachment to the shrinking vitreous. This explanation has been very strongly confirmed by Nordenson's investigations. There can be little doubt that it represents, if not exactly, very nearly what actually happens in many cases.

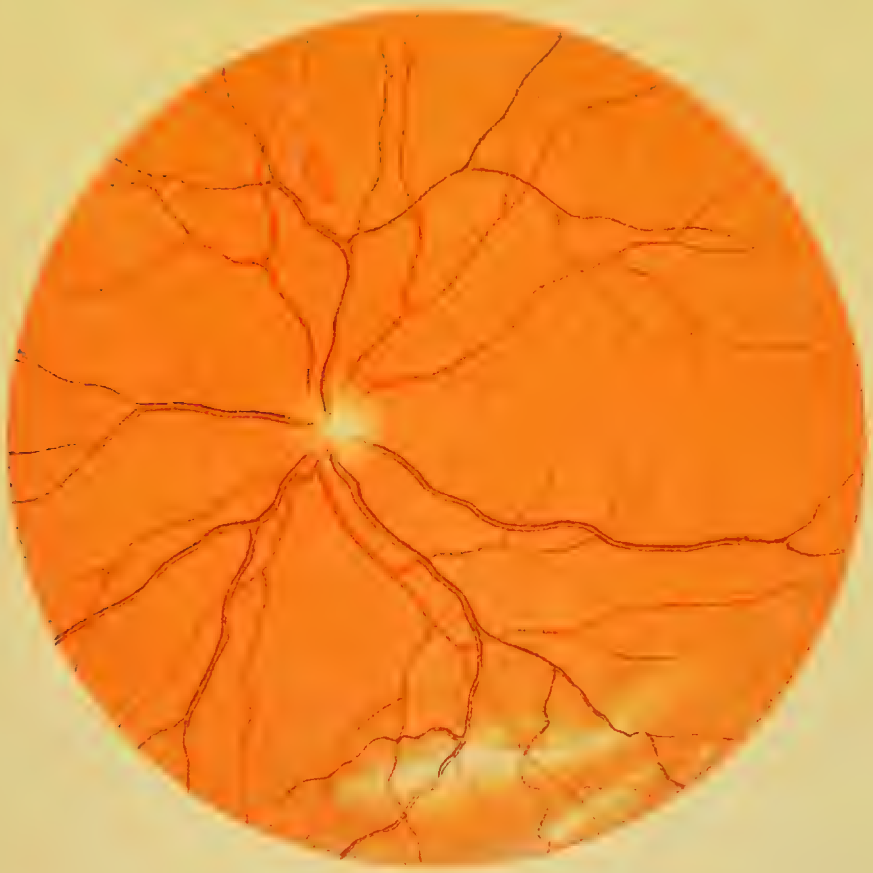

Fig. 64.—Case of detachment of the retina, with rupture.

A rupture in the retina would thus appear to be necessary for an idiopathic detachment to take place. As a matter of fact, such a rupture (see Fig. 61) is sometimes to be seen with the ophthalmoscope, but by no means invariably. In one hundred and nineteen cases in which Nordenson looked for it he found it only in forty-six. He remarks very truly, however, that the rupture, though present, might very readily escape detection with the ophthalmoscope, for a number of reasons—on account of opacity of the vitreous, by its being too peripheral, by being covered by a fold of retina, or owing to the reapplication of the portion of the retina which had been torn after a sinking of the fluid had taken place behind it. In the cases where it was seen it was found in all parts of the retina, but most frequently in the upper and outer quadrant. Only very rarely indeed was there more than one rent in the same eye. In some cases a deepening of the anterior chamber and retraction of the lens is observed along with detached retina, and is probably also due to shrinking in the vitreous.

The question naturally then arises, What gives rise to this consolidation and shrinking of the vitreous which appears from recent careful anatomical investigation to be an actual fact? The increase in the number of cases as age advances suggests the possibility of its being a senile change, and indeed the only difference in anatomical structure presented by the shrunken, as compared with the normal vitreous, is the greater density and approximation of the fibrous or connective tissue elements, suggesting a loss of more or less of the water by which they are in the healthy state swollen out. Whether this takes place without the occurrence of some form of choroidal inflammation, which gives rise to an interference with the nutrition of the vitreous, does not as yet seem quite clear. The greater frequency of the disease along with myopia is very probably due to the frequent association of choroiditis with that state of refraction, and not, as was first supposed by von Graefe,.to any dragging which the gradual distension of the elongated eye exerted on the retina. I have, for instance, seen detached retina in a father and two sons, aged ten and twelve respectively, the father with myopia, and the sons with some traces of choroiditis, but with emmetropic refraction. Here it is not perhaps too far-fetched to assume than the sons inherited that condition of the vitreous and choroid, which in the father led both to an antero-posterior elongation of the globe and a detachment from shrinkage, but in them merely to the latter. The occasional subsequent association with iritis in cases of detached retina points also to a uveal origin of the pathological condition of the vitreous, but it may well be that several causes exist for it.

The *treatment* of detached retina has hitherto been very unsatisfactory. The fact that in some cases the retina does become temporarily or permanently reapplied if the attachment be recent, suggests the advisability of keeping the patient for some weeks lying on his back. It is customary to combine this with pilocarpine injections, or with other means of promoting absorption. This I have generally done, but I cannot say that I have seen any permanent good result from it. Von Graefe, who believed that the cases in which he found a rent in the retina were more favourable, and who did not suspect any causal connection between it and the detachment, proposed and frequently carried out a perforation of the retina from within. This treatment was followed by others, until it was shown by Hansen Grut to be of no use. Removal of the subretinal fluid by puncture through the sclera has also often been tried, but without any marked success. An attempt was made some years ago by Wolff to revive this method of treatment, by which he claimed to have obtained remarkably good results. Unfortunately, however, others both before and since have not been able to confirm this. A constant drainage of the subretinal space by means of a piece of gold wire left sticking through the sclera was for some time tried by Wecker, also unsuccessfully. What is apparently wanted in the light of the present knowledge of the pathology of the disease is something which will prevent or counteract the shrinking process going on in the vitreous, and at the same time create a sufficient amount of adhesive inflammation between the choroid and the retina which has separated from it. Schöler claims to have obtained good results by injecting a drop or two of tincture of iodine into the vitreous. Those who have followed him in this treatment have not been able to confirm his statements with regard to it. So far as the method has as yet been developed it appears to be not only uncertain, but even dangerous. It may cause much more severe inflammation than is desired, and lead to destruction of the eye. One case has been recorded by Gelpke where it led to death from meningitis.

Detachment of the retina is, nevertheless, one of the few diseases of the fundus which we may not unreasonably hope some day to be able to check in some measure by active treatment. The more satisfactory knowledge which we now have of its pathology may possibly lead to some treatment which will prevent many cases from going on to complete loss of sight. It is evidently very desirable to discover some means of diagnosing at an early stage the detachment and shrinking of the vitreous which so often precedes it.

Opaque Nerve Fibres in the Retina.—As they pass through the lamina cribrosa of the optic nerve the fibres become divested of their myeline sheath. This accounts both for the comparatively small diameter of the papilla, and for the transparency of the fibres as they stretch over and form part of the retina.

In some cases, and in all probability as a congenital anomaly, because the myeline sheath is developed around the fibres of the optic nerve before birth, some of the fibres in the retina are rendered intransparent by being provided with a myeline covering. The appearance which this abnormality presents is very characteristic. It is seen as a brilliantly white patch, mostly extending from the upper or lower edge of the disc, sometimes from both at the same time. This patch almost invariably comes up to the margin of the disc, so as to appear quite continuous with it, while at its peripheral end it is less strongly defined, presenting a striated appearance owing to the sheaths of the fibres ending at different distances from their origin. The patch is rarely as big as the disc itself; when bigger, it curves round over the macula. Occasionally a detached portion, at some distance from the papilla, has been seen, in which case the nerves after having lost their sheaths have again regained them.

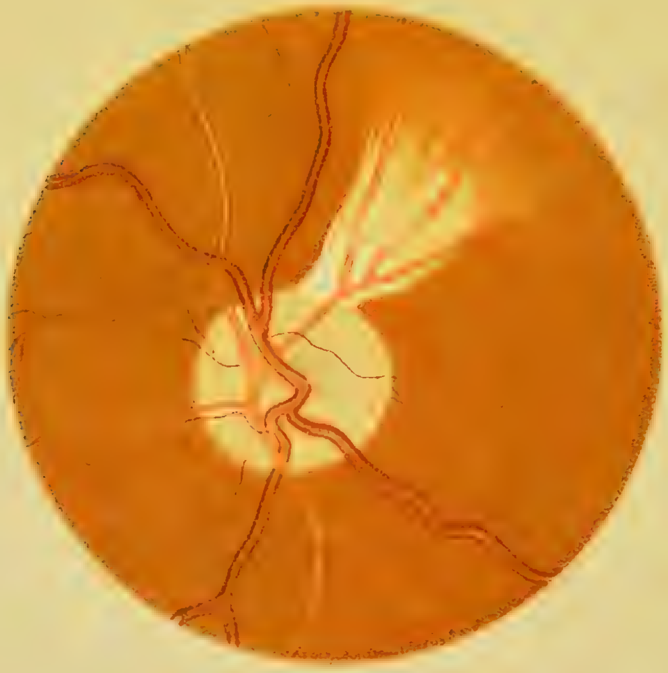

FIG. 65.—Opaque nerve fibres in the retina.

The vessels are almost always here and there hidden by the opaque fibres. Whether this is or is not the case depends partly on the depth and partly on the degree of opacity of the sheathed fibres.

Schmidt-Rimpler has shown in two cases that the sheath, though lost in the lamina cribrosa, is regained as soon as the nerves pass out into the retina. This is probably not always the case, as I have seen on several occasions densely white patches in the disc itself, which there could be little doubt were due to retention of myeline. In some cases, too, such irregularly shaped white patches exist in the papilla along with characteristic patches in the retina.

There is a little difficulty in diagnosing patches due to opaque nerve fibres (see Fig. 65) from inflammatory exudations in the retina. The latter are usually of a less dense white, they do not present the same striated appearance at their peripheral ends, do not come into such immediate contact with the margins of the disc, and are besides associated with hyperæmia, and often hæmorrhages, at other parts. I have seen the two conditions present at the same time without rendering the diagnosis difficult.

Opaque nerve fibres may occur in one or both eyes,—more frequently, I think, only in one. They do not give rise to any defect of vision, further than as a rule some corresponding degree of extension in the size of the blind spot. They are met with as the normal condition in some of the lower animals. In man they are uncommon, though by no means very rare.

RETINAL CHANGES PRODUCED BY STRONG LIGHT.—Very persistent after-images may be produced by looking at the sun or a strong electric light. If the exposure has not been too prolonged, the defect of sight thus caused may be recovered from. A prolonged exposure gives rise to a coagulation, or to some other unknown molecular change at the macula, with the appearance of a small irregular patch in that region, and the production of a persistent central scotoma. Less strong sources of light also give rise to macular changes. I have seen such apparently produced by constant use of the microscope. The changes in these cases appear to be more of the nature of small hæmorrhages at the macula. Sometimes they are apparently, in the less severe cases, mere temporary alterations in the arrangement of the pigment at the fovea. They may give rise to considerable central amblyopia with metamorphopsia, but the vision is usually slowly regained.

DISEASES OF THE OPTIC NERVE.

OPTIC NEURITIS.—Inflammation of the optic nerve occurs either alone, when it is often called *papillitis,* or along with more or less inflammation of the surrounding retina, giving rise to an appearance which goes by the name of *neuro-retinitis.*

A true papillitis is most frequently connected with some pathological change within the cranium; but such changes often lead to neuro-retinitis as well. It is characterised by more or less intense passive hyperæmia and swelling. The veins are engorged sometimes to an excessive degree, while the arteries, on the other hand, are normal in calibre or narrowed. The colour of the whole papilla is heightened, and the swelling causes it to become prominent, so that its surface is on a different level from that of the retina, while its margins, instead of being sharply defined, are more or less obliterated. The swollen nerve tissue also gives rise here and there to the disappearance of the vessels in the papilla, and these may be seen to dip abruptly at its margins as they continue their course in the retina. This obliteration of the margin of the disc is usually first noticed at its upper and lower edge, sometimes only to the nasal side. Often a very distinct striation can be made out on the swollen papilla as the bundles of swollen fibres are brought more prominently into view. There is at the same time more or less marked loss of transparency of the nerve tissue, and an increase, often considerable, in the apparent size of the disc. The degree of prominence of the swollen papilla can be appreciated by observing the difference in the strength of the glass, which is necessary to focus accurately the surface of the disc and the surrounding retina, and allowing roughly one millimetre for every three dioptres. The extent of parallactic movement, when on direct ophthalmoscopic examination the observer's head is moved from side to side, or when on indirect examination a movement is given to the convex lens in a plane parallel to the face, gives a very good idea of the intensity of the swelling. The amount of swelling met with is very variable, and the time taken for the development of these changes very different.

A considerable degree of papillitis may exist without interfering to any appreciable extent with the functions of the eye.

The central and peripheral vision for both form and colour may be perfect. Sometimes the absence of any subjective symptoms lasts for months, but as a rule it is not long before the visual acuity becomes more or less reduced, and the sight may indeed be altogether lost. When such loss of vision occurs pretty suddenly, it should always arouse the suspicion of there being some other cause for it besides the swelling in the papilla. The gradual loss of vision is no doubt due to compression of the nerve fibres in the papilla, by the gradual changes which the inflammatory exudation undergoes. An absence of visual disturbance at first affords no guarantee that such will not afterwards come on. When the central vision is affected, there is almost always at the same time to be found a more or less concentric limitation of the field of vision with greater or less loss of colour vision. Sometimes the restriction

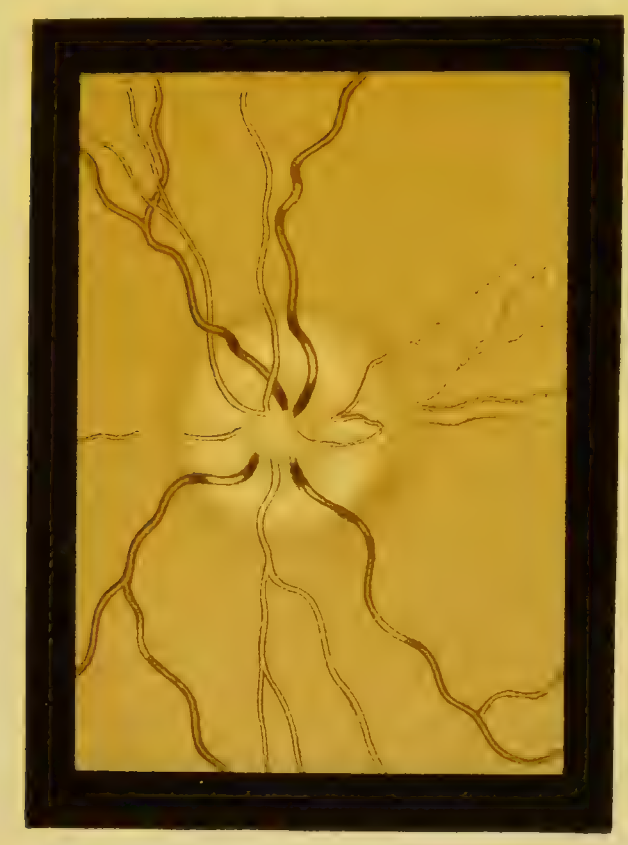

FIG. 66.—Early stage of optic neuritis.

of the field is more irregular, affecting often the nasal half of the field more markedly than the temporal. The blind spot may sometimes be found to be increased in size, especially if the test object be small and not very bright. The light sense is affected mainly in the way of appreciation of differences of illumination; there is no night blindness.

Encephalopathic papillitis, or papillitis symptomatic of disease in the brain, is practically always bilateral, although a few cases have been met with where it has occurred only on one side. Hughlings Jackson has recorded two such cases of unilateral optic neuritis, in each of which a tumour was found in the opposite cerebral hemisphere, and in which there was hemiplegia of the same side as the neuritis.

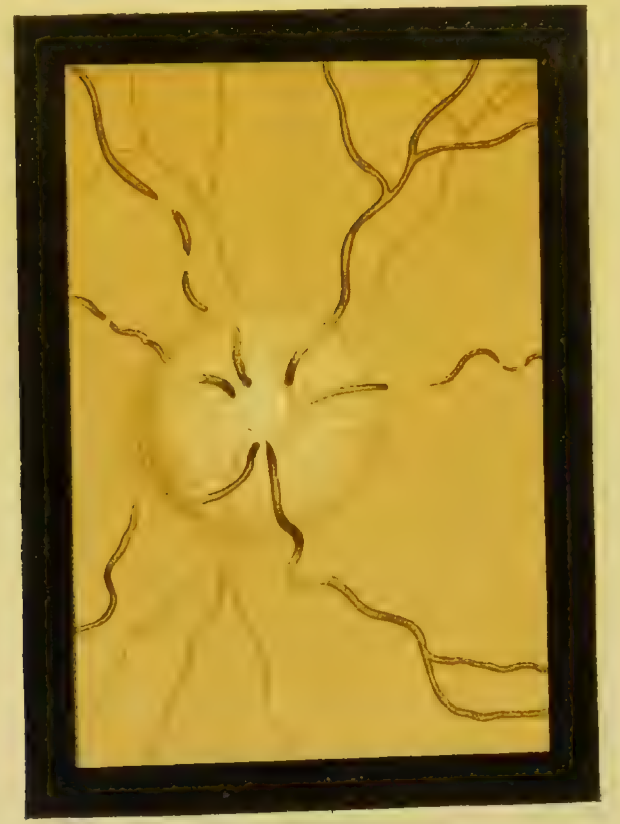

Fig. 67.—Later stage of optic neuritis.

The anatomical changes met with consist in the increase of the nuclei, and other evidences of inflammation, and generally in dropsy of the sheath.

The condition of swelling of the papilla in which an accumulation of fluid in the sheath is supposed to be present, is often called *choked disc.* It was at one time considered that an appreciable ophthalmoscopic difference existed between this condition and swelling due to descending neuritis, but it is now generally admitted that this distinction cannot be made clinically, and indeed there appears to be very little reason for even an anatomical classification of this nature. There are of course great differences in the degree of inflammation in different cases, but these differences are not due to essential differences in the etiology.

Not all pathological processes in the brain give rise to optic neuritis. It occurs most frequently with tumours; indeed, so frequent is this association, that its recognition must be looked upon as a factor of primary importance in the diagnosis of cerebral tumour. Next in point of frequency comes neuritis along with meningitis. Some cases of hydrocephalus are associated with optic neuritis. More frequently a simple atrophy of the nerves is found in this connection. It has been seen, too, in connection with purulent inflammation of the middle ear, when the inflammation has spread to the brain. I am inclined to think that this must be a very rare connection, as I have examined the eyes in a number of such cases without finding it. Papillitis is rare altogether in cases of abscess of the brain. It is not met with in cerebral apoplexies, or in softening due to thrombosis. Again, in other forms of cerebral disease with which it may in any particular case be associated, the question often arises, whether it is not more intimately connected with the primary disease of the blood or blood-vessels than with the manifestations of that disorder in the brain. I have, for instance, seen a case which was under the care of Dr. Affleck at the Edinburgh Royal Infirmary, in which there was pronounced optic neuritis, and in which, although death took place under the most marked "cerebral symptoms," the *post-mortem* examination revealed the entire absence of any brain disease.

A good deal of speculation, based on clinical as well as experimental facts, has for long been indulged in to account for the connection between optic neuritis and brain disease. The very frequent association with brain tumours, as well as the fact that neither the position nor the nature of the tumour in the brain seemed of special influence, not unnaturally led to the belief that the neuritis was intimately connected with the increased tension within the cranium which the growth caused, or might be supposed to cause. Von Graefe believed that this increased intracranial pressure caused compression of the cavernous sinus, and a consequent venous engorgement in the retina. This view, was, however, found to be untenable when the relation between the circulation within the eye and the brain was better understood, and it was found that there was such a complete anastomosis between the retinal and other veins.

Another hypothesis, generally known as that of Schmidt-Rimpler and Manz, then received support. According to this hypothesis, the cerebro-spinal fluid was driven along the sheath of the optic nerve, and caused compression or choking of the papilla, thus leading to more or less œdema and inflammation. This explanation followed shortly, and almost as a natural consequence, on the discovery made by Schwalbe of a direct communication between the arachnoid space and that existing between the optic nerve and its sheath. The distension of the distal end of the sheath has also been ascribed by Hughlings Jackson and Benedict to vasomotor changes, produced by irritation within the brain. It is difficult to see how such an irritation is to be ascribed to the action of a tumour which may be situated in any part of the brain. It is thus explained by Hughlings Jackson :—" I think that optic neuritis may be a doubly indirect result of local gross organic disease ; that, first, there are changes of instability about the tumour ; that, next, these lead on discharges by intermediation of vasomotor nerves to repeated contractions, with subsequent paralysis of vessels of the optic nerves or centres, and thus at length to that trouble of nutrition which is optic neuritis."

The distension of the sheath as an almost constant occurrence is an anatomical fact. Only a few cases have been recorded where it has not been found. That the distension is caused by fluid from the lymph spaces in the brain is at least extremely probable. The only weak point, then, in the choked disc hypothesis is the supposition that the pressure of fluid accumulated round the neck of the papilla should be capable of giving rise to the symptoms observed on ophthalmoscopic examination, or indeed to any true inflammation at all. The belief that the real or supposed pressure does not do so—a view in support of which Deutschmann has recently brought forward very strong experimental evidence—has led many to ascribe the inflammatory changes in the papilla to a descending neuritis. Thus Gowers says—" In a large number of cases of neuritis there is distinct evidence of a descending inflammation, either along the front of the optic nerve or along its sheath, and in cases of meningitis such descending inflammation is invariable. The distension of the optic sheath with serum has been regarded as the chief mechanism, but it is not essential for the production of neuritis ; it may be absent, and its occurrence is related especially to the presence of an excess of subarachnoid fluid."

It is not impossible that such a descending neuritis may have much to do with the production of the intraocular appearances in cases of meningitis. But not only is neuritis not nearly so frequent along with meningitis as with tumours, but in the latter connection the clinical appearances are greatly in favour of the papilla being more intensely inflamed than the trunk of the nerve. Some other explanation has therefore to be looked for. That given by Leber seems to accord more completely with all the clinical facts. Experiments made by Deutschmann under Leber's supervision, and more recently independent experiments by Scimemi, have led to results which greatly confirm Leber's views. Leber believes that the fluid accumulated at the distal end of the nerve sheath enters the lymphatic spaces of the nerve, and being of an irritating nature, there sets up inflammatory changes. In his own words, "Intracranial tumours, as also tuberculosis, give rise to congestion of the vessels, secretory inflammation, hydrops ventriculorum, and increased pressure. The products of tissue change of those neoplasms becoming mingled with the inflammatory transudations act as an inflammatory stimulus, and, passing with the cerebro-spinal fluid into the intravaginal space of the optic nerve as far as the eye, give rise there to neuritis and papillitis." Whilst, therefore, an inflammatory condition of the trunk may exist at the same time, owing to the same irritation by the transuded fluid, the tendency to accumulate round the papilla causes the more severe symptoms to be developed in that portion of the nerve, which is, to a great extent, visible with the ophthalmoscope. There is certainly nothing more extraordinary in this than the greater tendency towards inflammation of the anterior portion of the uveal tract in sympathetic ophthalmitis, although, according to the views now held as to its pathology, the substances giving rise to such inflammation must come in contact with other parts of the eye before reaching the ciliary body and iris. Evidently in both cases a certain accumulation of the irritating material, whatever its nature, must take place before marked inflammatory changes result. It has still to be shown that the fluid collecting in the nerve sheath is of a distinctly irritative nature, and if so, to what it owes its quality. The other symptoms accompanying the presence of tumours—headache, vomiting, &c., as well as certain anatomical changes which have been met with—undoubtedly point to the irritative nature of the exuded fluid.

There is one difficulty in the way of accepting Leber's hypothesis, viz., how are we to account for the very rare but undoubted cases of unilateral optic neuritis from cerebral disease? There appear to be at least three possibilities,—either they are only apparently connected with the process in the brain while actually due to some other co-existing cause; or, supposing such connection to be direct, they have a different and less common etiology—descending neuritis, for instance; or finally, while originating in the way indicated by Leber, some structural abnormality on the other side interferes with its manifestation there as well. One fact, which it may be of importance to remember, though possibly only a coincidence, is that the neuritis has been usually on the opposite side from that occupied by the tumour in the brain. The tumour might therefore well be supposed to exert sufficient pressure on the nerve on one side to prevent the fluid distension of its sheath. Such an explanation would be likely if the eye which was free from neuritis had nevertheless very defective sight.

There appears to be no invariable connection between the acuteness of the cerebral and ocular symptoms. According to Gowers, if the cerebral disease be acute, the accompanying neuritis is always acute. A chronic neuritis probably, therefore, signifies chronicity of the affection in the brain, while, on the other hand, an acute neuritis may be set up by disease of chronic as well as acute disease.

After existing for a longer or shorter time, the neuritic process as a rule passes on to atrophy of the nerve, with more or less complete destruction of sight. Most frequently the blindness thus produced is eventually complete, and this may occur long before the brain disease leads to the death of the patient. At other times, after having progressed to a certain extent, the process remains stationary, and some degree of sight is left. This, I believe, is more often the case in children than in adults, and probably mostly when the neuritis is originally connected with meningitis, in which cases it is generally less severe than when set up by tumour.

Some cases of neuritis get well without undergoing any, or only very slight traces of, atrophy. Mostly such cases have retained good vision throughout the period of inflammation, and only in such have I seen this practically complete recovery; but it appears undoubted that the vision, though defective at the time of the neuritis, may be restored without being permanently damaged by any subsequent atrophic process.

Tumours in the orbit, and inflammatory processes, which have either begun in the orbit or spread into it from surrounding parts, may give rise along with proptosis to optic neuritis. As a rule, the swelling in the nerve is not so intense as in cases connected with tumours in the brain, and often a considerable acuity of vision may be retained, notwithstanding a high degree of protrusion. Deformities of the skull, owing probably to pressure on the nerve at the optic foramen, may be the cause of neuritis. Since this connection was first pointed out by von Graefe, a good many cases have been recorded. Nettleship and a few others have also observed post-papillitic atrophy in cases of congenital hyperostosis.

A number of cases of neuritis are altogether unconnected with disease either in the cranium or orbit. The etiology in such cases is often not very easily made out. They may occur after severe illnesses without there being any definite connection between the particular disease and the neuritis. Other nerves may be affected at the same time; it is found, for instance, as one of the symptoms of more generalised neuritis. Syphilis, exposure to cold when there is more or less of a rheumatic disposition, and menstrual disturbances (particularly amenorrhœa and sudden suppression) all appear to be of undoubted causal influence. In all such cases, and especially in the more acute forms, the prognosis is on the whole much better than where the neuritis is merely a secondary manifestation. Even in cases where almost total blindness has been produced by the inflammation, a complete, or nearly complete, recovery may take place. Often a high degree of amblyopia, with or without restriction of the field of vision, is left.

An interesting and peculiar clinical type of optic neuritis is one which comes on generally shortly after puberty, almost exclusively in young men, and without there being any apparent immediate cause, other than a more or less markedly neurotic temperament. In such cases there is usually distinct evidence of heredity, several members of a family being affected. It always occurs in both eyes, causing generally pretty suddenly considerable blindness, first of the one eye and then of the other. The central vision is sometimes so much destroyed as to give rise to an almost absolute central scotoma from which the patient does not as a rule recover. The objective symptoms are much like other cases of neuritis,—they are rarely very pronounced, but there is usually well-marked perivasculitis.

The pathology of these cases is probably very closely allied to a not by any means uncommon, yet not very sharply defined, group of cases, which are generally looked upon as of the nature of *retrobulbar neuritis.* The characteristics of this group are,—very slight changes in the papilla, and the presence of a more or less definite central scotoma. The changes in the papilla may be so slight as altogether to escape detection, or they may amount to merely some slight haziness of its margins, and perhaps a trace of perivascular inflammation. After some time the outer half of the disc may exhibit an abnormal degree of pallor, whilst at the same time the contrast between it and the nasal half may be increased by some degree of injection of the latter. At other times a more general pallor of the disc results.

The central scotoma has often very much the same form as that which characterises toxic amblyopia, but it may differ from it in an important manner both in shape and extent. Just as in these cases, it is usually negative,—that is to say, it does not give rise to the sensation of any dark spot. Often a faint cloudiness is complained of in a bright light, and on account of this patients frequently state that they see better in a subdued light. On examination, however, this does not prove to be the case. At other times a more or less dark, generally buff-coloured, mist is complained of. The colour defect, too, in the region of the scotoma is much the same as in the toxic amblyopia. The scotoma does not present, however, the same regularity of shape, and is not confined to the area stretching from the disc to the macula to that area which is supplied by the so-called papillo-macular fibres, changes in which have been recently demonstrated by Nettleship, Uhthoff, and others to exist in certain cases of toxic amblyopia.

A point of considerable importance is the frequent extension of the affected area considerably to the inner side of the point of fixation, a condition which is not met with in typical cases of toxic amblyopia. At the same time in many cases there is some concentric limitation of the field of vision. Both eyes may be affected, but much more frequently only one. Where the disease is bilateral it rarely gives rise to the same degree of amblyopia in the two eyes. It is about equally common in men and women.

The disease with which it is most likely to be confounded, and with which pretty recently it has been confounded, is the much more common toxic amblyopia. This mistake is not likely to be made except in bilateral cases. Even in one-sided cases the loss of sight of the other eye from some different cause, as I have indeed several times seen, may lead to this difficulty. The points to be attended to in the differential diagnosis are mainly,—the irregular shape of the scotoma, more particularly its extension when large to the nasal side of the point of fixation, and the restriction of the field of vision. In some cases there is more or less pain complained of on moving the eye, or produced by pressing the eye back into the orbit. Generally, too, the pain is most marked when the eye is moved in some particular direction.

The *prognosis* is much less favourable than in toxic amblyopia. Some cases go on to complete blindness, others are left with an absolute central scotoma and the ophthalmoscopic appearances of optic atrophy. A certain proportion —not quite half probably, and these the more acute and less severe cases—recover in a few weeks from the onset of the disease.

There can be no doubt that the symptoms are produced by a superficial inflammation of the nerve in the orbit, an inflammation which for some reason or other has a greater tendency to affect, and probably also to extend deeper into, its temporal portion. Very probably, as Nettleship has suggested, the differences met with in different cases are to some extent due to the distance of the focus of inflammation behind the eye. This supposition would not only account for the differences in ophthalmoscopic appearances, but also, as the arrangement of the fibres supplying the same parts of the retina differs at different distances along the course of the trunk, for differences in the symptoms as well.

In the *treatment* of optic neuritis the cause has in the first place to be taken into consideration. Some cases depending on intracranial tumour have been relieved by the removal of the tumour. Much progress has been made recently in this department of surgery. As far as any possible effect of such treatment on the vision goes, it would have of course to be undertaken before the inflammatory changes in the nerve had proceeded too far. Removal of the fluid from the sheath of the optic nerve was recommended by Wecker as far back as 1872, but has not received much attention. It has lately been taken up by Carter, and from the results obtained by him it seems possible, taking into account the probable pathology of the neuritis, that an operation for this purpose may prove to be of some value in preventing complete blindness. Some physicians, whose authority in the matter must be acknowledged, are very strongly in favour of treating all cases of optic neuritis in the first or preamaurotic stage with iodide of potassium.

ATROPHY OF THE OPTIC NERVE.

Atrophy of the optic nerve may be *primary*, that is to say, it may originate in the nerve itself, or it may be *secondary* to changes which have occurred either in the retina or in the central nervous system. The atrophy may, as we have seen, follow inflammation; it is then usually termed post-neuritic or post-papillitic atrophy, or it may be simply due to degenerative processes of different natures.

The ophthalmoscopic appearances are—pallor of the disc, often accompanied by some degree of excavation, and by narrowing of the calibre of the vessels. The scleral ring is generally more than usually well marked, owing no doubt to some degree of shrivelling or contraction, which only rarely gives rise to any distinct diminution in the size of the disc. The loss of colour in the disc, which first becomes marked as a rule to its outer side, is due partly to diminution of vascularity, and partly to disappearance of the nerve tissue, leaving only the white connective tissue, often increased in amount, behind. When this is the case, there is produced a shallow excavation known as an *atrophic excavation.*

From the mere colour of the disc, without taking into consideration the size of the vessels and the existence of an excavation of this nature, it would be rash to conclude that there was atrophy, unless perhaps the light reflected from its surface had not only lost all tinge of yellow or pink, but also become bluish or green in hue. Considerable variations take place with respect to colour within physiological limits; a very anæmic condition of the disc may, too, although it may hardly be normal, exist without any, or any great, functional disturbance, and yet not be of the nature of atrophy. Great pallor, with loss of transparency of the disc, due to consolidation of some exuded matter, is found in cases in which a neuritis has been completely recovered from, and not followed by atrophy. A similar white intransparent appearance is given by the presence of myeline in the nerve fibres of the papilla, a condition occasionally met with in the healthy human eye, and one which is the rule in some animals.

Even when the ophthalmoscopic appearances, pallor, excavation, and narrowing of the vessels leave no doubt as to the existence of an atrophic process, it is impossible to say, with any degree of certainty, from a consideration of the picture thus presented to one, whether the process is a progressive one or not. When the atrophy is evidently post-neuritic, the chances of its becoming arrested at some stage short of complete blindness are generally greater than when it is the result of a more distinctively degenerative change. Post-neuritic atrophy is characterised by more or less indistinctness and want of definition of the margins of the disc, and in many cases by white patches on its surface. These patches stretch between, and sometimes cover, the bifurcations of the vessels, and extend along the main trunks as white lines. This condition of perivasculitis is often not confined to the disc alone, but extends for a greater or less distance along the vessels into the retina.

A good deal more light is often thrown on the question as to whether in any given case the atrophy is likely to be progressive or not—that is to say, is likely to progress to such an extent as eventually to destroy vision—by a careful examination of the subjective symptoms caused by the degeneration. These symptoms are—defects in the central vision, restrictions of the field of vision, and defects in the colour and light sense.

In all cases where the atrophic process in the nerve is primary, or due to some interruption at any part of its course from the papilla to its termination in the brain, and not secondary to alterations in the choroid and retina, there is no interference with the light sense in the way of appreciating small degrees of illumination,—that is to say, there is not true night blindness. On the other hand, the power of distinguishing between intensities of illumination is less acute than normal, often indeed very markedly so. Such a defect of the light-sense is characteristic of any affection originating in, or mainly implicating, the nerve tissue itself.

The most common form of restriction of the field in atrophy is the concentric. Often, however, the defect in one direction may be much greater than in others, and when this is the case it not infrequently happens that a good deal of symmetry in this respect is to be found in both eyes, even although the atrophy may be much more advanced in the one than in the other.

In all cases of progressive atrophy, the restriction which is taking place can probably be most easily demonstrated for colours. This is mainly because, if the examination be made in ordinary daylight, the corresponding failure of the sense of form may, if slight, more readily escape detection. If the test be made by Bjerrum's method with very small objects (see Chap. I.) the restriction for form can always be demonstrated. There seems every reason to suppose that the colour defect in progressive atrophy is accounted for by the mechanism for the conveyance along the nerve of physical stimuli destined to give rise to sensations of colour, being one necessitating a higher order of differentiation than is the case with the sense of form.

Restriction of the field of vision, when more or less concentric, and especially when the colour defect is pronounced, is always very suggestive of progression.

Usually the defect of central vision keeps pace with the gradual narrowing of the field, so that when there is much restriction there is generally at the same time considerable amblyopia. In some cases the central vision remains relatively very good, but it is seldom that this can be looked upon as a good sign,—in fact, the prognosis mainly depends on the state of the peripheral vision. In the same way, if during the progress of a case it is found that the vision gets no worse for some time this must not be taken as a good sign, unless at the same time the state of the field remains stationary as well. Some cases, particularly when the atrophy results from neuritis, progress to a certain extent, leading to both restriction of the field of vision and defective central vision, and then become arrested.

In all cases where there is merely a central scotoma without any narrowing of the field, the prognosis is good,—that is to say, the scotoma may become absolute, but vision is not likely eventually to be lost altogether, notwithstanding the objective appearances.

Neither from the ophthalmoscopic changes nor from any peculiarities in the symptoms does it seem possible to diagnose the cause of the atrophy without taking into consideration other circumstances. An atrophy due to spinal disease is often, though not always, complicated at the time of examination by ataxic or other spinal symptoms, or by abnormalities in connection with the pupil, loss of knee-jerk, &c.; one which is connected with cerebral disease is frequently followed, though rarely preceded, by mental derangements. Some of the primary degenerative changes in the optic nerve, which are always bilateral, are local expressions of the same conditions which give rise to tabes, and as such may precede the other symptoms. They are not extensions, but independent degenerations of probably exactly the same nature as those which Pierret has demonstrated frequently exist in the cutaneous nerves. Sometimes the degeneration begins at the central terminations, at other times at more peripheral parts of the optic nerves. Apart from what has been shown to exist anatomically, Nettleship considers that the absence of pre-atrophic amblyopia affords strong clinical evidence of the peripheral and entirely isolated nature of the degeneration, which in cases of tabes leads to progressive atrophy. In other cases it appears to be altogether unconnected with any similar process elsewhere, or are at all events not, even after the lapse of many years, followed by any changes in the central nervous system, even although they may perhaps be looked upon as not improbably manifestations of the same disease.

Unilateral atrophies are generally the result of some process originating either in the retina or the orbit, but they may be caused by compression within the cranium either of the nerve or the vessels supplying it. The latter cause may be suspected when there is marked evidence of arterio-sclerosis elsewhere. Of the bilateral forms of cerebro-spinal origin some idea of the process giving rise to them may be gathered from the ophthalmoscopic appearances; thus the opaque-looking atrophic degeneration, with narrowing of the vessels, points to changes mainly involving the connective tissue and destroying the nerve elements. It may be taken as a general rule—to which, however, there are exceptions—that degenerative processes beginning at the base of the brain, while they lead to pallor, are not associated with diminution in the vessels unless the atrophic process has at any time been complicated by a papillitis. Processes which begin in the papilla itself, and involve the connective tissue, as they affect that portion of the nerve in which the central vessels are situated, do cause constriction of these vessels, and thus gave rise to a different ophthalmoscopic picture.

A large proportion of cases of bilateral atrophy are certainly due to the same causes which give rise to degenerative changes in the sensory tracts of the spinal cord and brain. What this proportion is cannot well be ascertained. Nettleship's statistics give fifty-eight out of seventy-six cases, or fully seventy-five per cent. This is possibly rather under than above the true amount, as a great many cases are not seen by ophthalmic surgeons at all, but are recognised by the physicians by whom the patients are treated for their other symptoms. Indeed, this proportion of the cases referred to is only that in which some undoubted signs of disease of the central nervous system existed; in ten cases more, such a connection was more or less probable; this would raise the percentage to about ninety, which may be taken as not far off the mark. Uhthoff has also given statistics of one hundred cases of atrophy. From these, however, must first be excluded seventeen depending rather on neuritis or some orbital affection, &c. Of the eighty-three remaining, sixty-nine, or eighty-three per cent., were connected with some more or less evident spinal or cerebral disease. The percentage of cases of tabes dorsalis, in which optic atrophy shows itself at some time or other, is estimated at from fifteen to twenty per cent. Atrophy also occurs in disseminated insular sclerosis and in lateral sclerosis, but is not met with in affections of the nuclei of some of the cranial nerves.

The ophthalmoscopic changes characteristic of atrophy are not marked where it is due to disease at one visual centre, and the defect in vision produced is of the hemianopic type, yet it is known that degeneration does spread downwards in such cases. Possibly affections of both hemiopic centres, or of other visual centres, supposing such to exist, may be the cause of some atrophies which extend far enough to become visible with the ophthalmoscope. It seems likely that when, as happens in a certain proportion—probably quite ten per cent. of cases of general paralysis—there is optic atrophy, there is at the same time some spinal degeneration. Atrophy in disseminated sclerosis is most frequently accompanied by other paralyses of cranial nerves, *e.g.*, of the sixth, seventh, &c.

The eye symptoms in connection with spinal lesions are often of importance from a diagnostic point of view, as showing the eventually slow degenerative nature of the disease, even when, as sometimes happens at the time of first examination, the symptoms from the side of the cord are acute. Spinal as well as primary optic atrophy is a great deal more common in men than in women.

Altogether, optic atrophy has been found to account for about one-fourth of all cases of blindness. As one pretty fruitful source of blindness, viz., ophthalmia neonatorum, is now less active owing to better prophylactic measures being taken than formerly, the percentage of cases of blindness due to atrophy may be even higher than this.

CHAPTER VII.

DISEASES OF THE IRIS AND CILIARY BODY.

The Iris.—Notwithstanding that a most intimate anatomical connection exists between the different parts of the uveal tract (the iris, choroid, and intervening ciliary body), it is more common to find inflammation localised either throughout its whole course, or, for some time at all events, to one particular portion of that tract, than to meet with a general uveitis.

This is greatly owing no doubt to the differences which exist in the source from which the blood supply for the various parts is immediately drawn. In this respect the iris and ciliary body are more intimately connected than is either with the choroid, both being supplied by the anterior and posterior long ciliary arteries. The choroid, one of whose functions is to convey nutrition to the other parts of the eye, is not only supplied from a different source, viz., the short posterior ciliary arteries but is traversed by a network of vessels which are relatively richer in blood.

The choroid, with its peculiar vascular supply, is most subject to inflammation occurring in variously disposed and localised patches, except in the case of the hæmorrhagic and suppurative forms of inflammation. The anterior portion of the uveal tract shows, on the other hand, a greater tendency to diffuse inflammation. The iris and ciliary body are frequently inflamed at the same time. This is almost invariably the case when the inflammation is first set up in the ciliary body. When originating in the iris it is not uncommonly either entirely or mainly confined to it.

Inflammation of the iris—*iritis*—may, from a clinical point of view, be looked upon as occurring under three forms—(1.) plastic; (2.) serous; (3.) purulent. This classification, according to the nature of the secretion from the inflamed tissues, is justified not only on account of the types themselves being tolerably distinct types, and leading, apart from complications, to different changes within the eye, but also on account of the difficulty of establishing any satisfactory classification based on the etiology of each case. It is of course of the utmost importance to consider the etiology as well, yet it is only rarely that this can be made out by an examination of the ocular changes alone.

Iritis may be *primary*, or *secondary* to some general affection or dyscrasia. The most important forms, considered etiologically, and pretty much in the order of frequency in which they are usually met with, are—(1.) rheumatic, (2.) syphilitic, (3.) traumatic, (4.) sympathetic, (5.) gonorrhœal, and (6.) tubercular, iritis.

HYPERÆMIA OF THE IRIS.—Congestion of the vessels in the iris, which gives rise to some discoloration of the iris as a whole, and often at the same time to more or less contraction of the pupil (myosis), is a frequent accompaniment of irritation or inflammation elsewhere in the eye. It is met with, for instance, very frequently where foreign bodies, lodged in the cornea for some time, have produced a local irritation. It is also seen along with different forms of keratitis, with scleritis and inflammation of the deeper coats of the eye, and is, of course, also one of the first changes which take place when the iris itself becomes either primarily or secondarily inflamed. The hyperæmia gives a greenish coloration to blue, and a dirty reddish appearance to dark irides.

When myosis—what has been by Nettleship very appropriately called *congestive myosis*—exists along with the hyperæmia, the pupil should be kept dilated with atropine.

IRITIS.—Inflammation of the iris, from whatever cause, presents certain well-marked symptoms. When acute, there is always considerable *pain* referred not only to the eye, but also, and in most cases indeed principally, to the surrounding parts—forehead, sides of the nose, &c. The pain is usually much less severe in the daytime—exacerbations coming on at night and during the early hours of the morning. In the serous and chronic forms of iritis there is generally very little pain, sometimes practically none—not sufficient, in fact, to direct the patient's attention to the changes taking place.

The pain of iritis is spontaneous, and accompanied by photophobia and lachrymation. It is often increased by light falling into the eye, and also by attempts at reading or using the eyes for any purpose requiring active accommodative effort—in short, by anything which produces, or tends to produce, changes in the shape of the pupil, and thus interferes with the state of rest of the iris. Temperature, too, is not without influence. As a rule, the pain is increased by cold, whereas warmth has often a markedly soothing effect. There is, however, little or no pain on pressure in uncomplicated cases, unless they be very severe: tenderness to pressure indicates, as a rule, a complication with inflammation of the ciliary body (cyclitis). The course of an acute iritis is, in fact, pretty definitely indicated by the amount of pain. As the inflammation subsides the pain becomes less severe, while greater pain at any time almost invariably co-exists with exacerbations of the inflammation. As a rule, too, the more acute the pain the more severe is the inflammation.

Another constant symptom of iritis, though by no means pathognomonic of it, is a *circumcorneal injection*—that is, an injection of the fine network of deeper vessels which immediately surround the cornea. These vessels, the episcleral or subconjunctival vessels, are branches of the anterior ciliary arteries and veins. They form a network around the cornea, a quarter of an inch or so in breadth, the finest meshes of which lie nearest to the cornea. According to the severity of the iritis, there are differences in the breadth of the ring of injection. Less severe cases are accompanied by injection of only a comparatively narrow band, *i.e.*, only the vessels immediately surrounding the cornea are distinctly visible as a pinkish zone. With increased intensity in the iritis this zone widens out, and in very severe cases may be associated with hyperæmia of the more superficial vessels of the ocular conjunctiva as well, or even in the most acute plastic forms, and in purulent iritis, with chemosis. When there is no irritation or focus of inflammation in the cornea, a circumcorneal injection is indicative of a deep-seated inflammation, so that its existence in such cases should at once direct attention to the iris.

The breadth and intensity of the circumcorneal injection is, just as the amount of pain suffered, a good measure of the severity of the iritis. With ordinary care it is impossible to mistake it for conjunctival hyperæmia, but the possibility of the deep and superficial injections co-existing should be borne in mind, as in that case the differential diagnosis presents rather more difficulty. It is seldom that any marked degree of swelling or redness of the eyelids occurs in iritis, except in the purulent form; at most, there is usually only a little redness confined to the margin of the upper lid.

A third symptom is *hyperæmia of the iris.* The hyperæmia is often most distinctly visible in the region of the sphincter pupillæ, *i.e.*, in the zone almost immediately surrounding the pupil, where some of the branches of the arteries in the iris form a ring. Sometimes, commonly enough in cases of traumatic iritis for instance, the engorged vessels give way and lead to an effusion of blood into the anterior chamber (hyphæma). The inflamed iris is not only altered in colour, owing to hyperæmia, but the glistening appearance which its surface presents in health is lost, while the markings on its surface, in so far at least as these can be seen with the naked eye, are more or less effaced. This *muddiness of the iris* is the result of exudation into the surrounding aqueous, as well as into its own tissues, and is therefore more or less complete according to the nature and amount of the exuded material. The parenchymatous exudation leads to thickening of the iris. The thickening is usually not very great, and tolerably uniform throughout, although perhaps most marked near the pupil. Sometimes, however, it forms prominent masses projecting more or less into the anterior chamber, and single or multiple. Such masses are mostly either tubercular or syphilitic in their nature.

The exudation, which does not infiltrate the tissues of the iris itself, passes into the posterior and anterior aqueous chambers, and leads to attachments, more or less complete and firm, between the posterior surface of the pupillary margin and the anterior capsule of the lens. These attachments, or *posterior synechiæ* as they are called, form one of the chief and most serious complications of iritis. They interfere with the mobility of the pupil, and render the result of subsequent attacks much more doubtful than they might be, as not only do fresh synechiæ then form, which might otherwise be prevented, but the pupil may be more or less occluded by exudation passing from one side to the other. The synechiæ which form in an ordinary case of iritis, where the inflammation has not been prolonged and severe or the exudation excessively plastic, are generally punctiform. That is to say, there is not, as a rule, from one attack, and more especially if proper treatment has been adopted at an early stage, a continuous attachment of the pupillary border to the lens capsule ; only isolated attachments here and there. These, too are often most numerous at the lower part of the pupil, as the exudation which gives rise to them gravitates to the most dependent portion of the area over which the back of the iris is in contact with the lens capsule, and this is most often the lower part of the pupil. They are apt to form during the night, partly on account of the exudations which then take place, and partly, no doubt, owing to the iris then being more constantly at rest than during the day. The tacking down of the iris is not noticeable, as a rule, unless an attempt is made to dilate the pupil, when, instead of being round, it assumes an irregular shape, due to irregularity in the dilatation which results from adhesions in some situations and not in others. Some days elapse before the synechiæ become so firm as to resist any ordinary effort, such as may be made by a mydriatic, that is, a substance which has the power of causing dilatation of the pupil. Fresh attachments yield in this way, usually leaving behind them some pigment from the back of the iris, so that it is not an uncommon thing to see, in an eye in which there has previously been iritis, a number of pigment spots on the surface

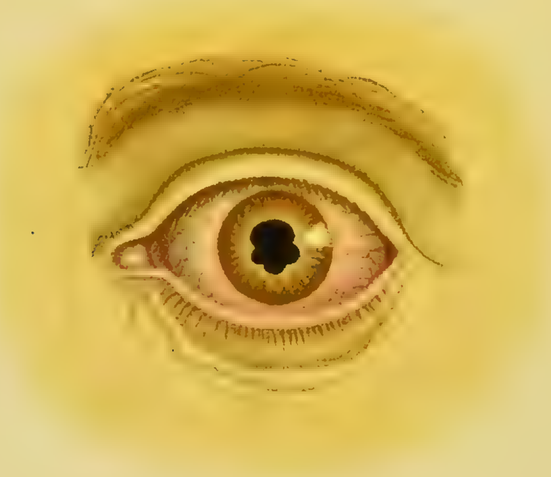

Fig. 68.—Irregular pupil owing to synechiæ, from a case of iritis under atropine.

of the capsule, indicating the positions of the former attachments.

In severe cases of iritis, or in cases where proper treatment has been neglected at the first, and in which there have been more or less frequent recurrences of inflammation at different periods, a complete agglutination between the iris and capsule takes place. Sometimes this may be so complete as to abolish altogether the space through which the aqueous humour which is secreted from the walls of the posterior chamber finds its way into the anterior chamber. This is called *exclusion* of the pupil. In the very worst cases, always complicated with cyclitis, the exudation is extremely plastic and copious, and leads not only to agglutination of the pupillary margin, but to an attachment of the back of the iris to the lens capsule, and to a practical obliteration of the posterior aqueous chamber. The complications which arise from these conditions are afterwards discussed.

The exudation which passes into the anterior chamber causes in many cases a mere cloudiness and discoloration of the aqueous humour, more or less marked according to the amount existing at the time of examination, and generally denser at the most dependent part of the chamber. In the so-called serous iritis little deposits of lymph take place on the walls of the anterior chamber.

In some cases of plastic iritis a spongy-looking, fibrinous mass may be seen lying in the anterior chamber. This, too, occasionally consolidates into what bears a very striking resemblance to a dislocated clear lens in this position. So close, indeed, is the resemblance, that if the case is then seen for the first time, the diagnosis may be by no means easy. In purulent iritis the pus secreted from the inflamed tissues collects in the anterior chamber, where it falls to the most dependent part as *hypopyon*.

The hyperæmia of the iris, as well as the synechiæ, give rise to another symptom of iritis, viz., a *sluggishness in the movements of the pupil*, so that if the eyes be alternately covered with the hand or lids and exposed to the light, the dilatation and contraction which should then take place are much less marked than under the normal conditions. This symptom affords a means of rapidly ascertaining whether, when there is great injection of the conjunctiva, it is due to conjunctivitis alone, or conjunctivitis complicated with iritis. This simple test should always be made if the other signs of iritis are not present.

In uncomplicated iritis the vision suffers at first only in so far as can be accounted for by the amount of obscuration in the pupil and aqueous humour in front of it. There is therefore in such cases frequently no great *diminution of visual acuity*. In cases where there is, the pupil is to a great extent occluded by lymph, or the iritis is complicated with choroiditis, or with cyclitis, along with effusion into the vitreous. Before referring any visual defect found on examination to the inflammatory changes in the eye, it is well to test whether vision is not improved with any glass. Owing to the inflammation a previously latent degree of hypermetropia may become manifest, or, on the other hand, there may be an abnormal degree of spasm of the ciliary muscle from irritation, giving rise to myopia. It occasionally happens, too, that the lens is somewhat pushed forward, so as to give rise to a slight degree of myopia.

Some *increase in intraocular tension* probably always takes place in iritis. When there is not a very copious exudation it is often, however, impossible to detect the increase; in other cases, especially in serous iritis, it may be very marked indeed. At later stages of the disease, after the supervention of secondary changes, it is very common to find first an increase, and subsequently a diminution of the tension within the eye.

Very great differences are met with in the *duration of an attack* of iritis, as many cases, even such as eventually lead to the very worst consequences, as far as the functions of the eye are concerned, run a chronic and more or less painless course from the beginning. In some more acute cases, again, relapses may take place, and thus the inflammation be protracted beyond its natural duration. Very much, indeed, depends on the treatment adopted from the first. Most acute cases, if properly treated, last from three to six weeks, the duration being more frequently nearer the lower limit than the higher.

In some cases, where the iritis exists along with some other affection, as for instance some cases of gonorrhœal iritis, in which there are mostly manifestations of gonorrhœal rheumatism at the same time, there is a tendency to the recurrence of the iritis every time that the symptoms with which it has previously been associated make their appearance. But there is an important class of cases in which the recurrence takes place from time to time without there being any very marked association with other affections. Such cases have received the name of *recurrent iritis.* Sometimes the recurrence takes place at regular intervals, most frequently at or about the same season, year after year. More commonly there is no such regularity exhibited in the periods of recurrence.

It has long been a disputed point as to how far the presence of synechiæ leads to recurrent attacks of inflammation of the iris. Some maintain, for instance, that the dragging on the iris, which the synechiæ occasion when the pupil responds actively to changes in the amount of light, is the main, if not the only cause of such recurrence. Others again deny any influence at all from the side of the synechiæ. Many eyes in which synechiæ are present are not subject to recurrent iritis, while on the other hand recurrent attacks are met with in cases where there are no synechiæ. It has been observed, too, that where any association with other rheumatic affections—for it is mainly if not entirely in the rheumatic form of iritis that recurrence takes place—is found to exist, the repeated attacks are connected more or less intimately with other manifestations of the same constitutional weakness. It seems probable, therefore, that recurrent iritis is as a rule, at all events, not due primarily to the synechiæ. Still the presence of the synechiæ, by interfering with the proper dilatation of the pupil, tends to prolong the attacks.

When, instead of isolated or discontinuous attachments, an annular adhesion has taken place, so that the pupil is completely blocked, further changes usually soon begin in the eye, owing to the fluid secreted from the posterior chamber not being able to find its way into the anterior chamber, and thus escape from the eye. The iris is then bulged forward, so that the anterior chamber is shallowed, except in that portion corresponding to the pupil, *i.e.*, at its centre. The appearance presented by this condition is well called by French writers "iris bombé." The vision then no longer corresponds to the opacities in or in front of the pupil, but is diminished by changes in the vitreous or choroid, or more frequently by those which the accompanying increased intraocular tension gives rise to. Eventually complete blindness from detachment of the retina, or secondary glaucoma, is the result in such cases, if operative interference has not been made in good time.

Iritis is most common during the first three decades of life, and perhaps least common in infancy or early childhood. It occurs, as Hutchinson amongst others has pointed out, as a rare manifestation of inherited syphilis in very young children, mostly before the end of the first year; sometimes, indeed, during intra-uterine life, and at a later stage when associated with interstitial keratitis. In older children the serous form is practically the only one met with, if we exclude traumatic and sympathetic iritis and the rare tubercular inflammation of the iris.

The *treatment* of iritis should vary to some extent according to the cause of the inflammation, and may therefore be considered under the headings of *special* and *general* treatment. The special treatment, applicable to the different forms of iritis, is referred to in the connection with the discussion of each form. The general treatment, applicable to all cases, may be divided into that to be adopted at the time of the inflammation, and that which is necessary in order to avert the consequences of the complications to which it gives rise. What should be aimed at, at the time of the inflammation, is to put the eye as far as possible at rest, to prevent the formation of synechiæ, and alleviate the pain.

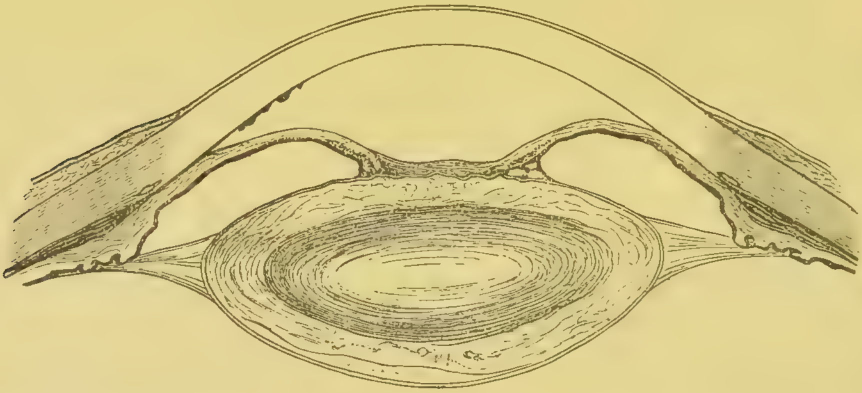

FIG. 69.—Iris bombé shown in section. After Fuchs.

The patient should wear either darkened glasses—the best kind being what are called "London smoke" glasses—or have both eyes well covered by a shade. The shade should come well down over the eyes at the sides as well as in the front, and while thoroughly keeping the light away, should be not too heavy. One of the best materials for such a shade is ordinary brown paper. It is not necessary in most cases, if this precaution be taken, to keep the patient in a darkened room. He should not, however, be in a very strongly illuminated one. All attempts at reading or writing must be stopped. Owing to the association which exists between the impulses to pupillary and accommodative movements in the two eyes, it is impossible to comply properly with the indication for rest, unless both eyes be kept from sudden and constant changes in illumination, or from efforts at accommodation; hence it does not suffice to shade one eye alone, or to allow the patient to read with the sound one while the other is bandaged, as is often done. An attempt must be made to get the pupil thoroughly dilated with atropine, and this dilatation should be kept up as long as any circumcorneal injection lasts. Care should be taken in applying the atropine drops that they really get into the eye, and are not washed away by the copious flow of tears met with in most cases. When atropine is not tolerated, duboisin or daturin may be substituted.

If the case has first come under observation when the synechiæ have begun to form, continued use of atropine may lead to their rupture, and a good dilatation may be got even after several days.

In most cases, at all events, the unattached portion of the pupil dilates, and further mischief is to a great extent obviated. Where, however, there are not only dense but numerous attachments already formed, and a few days' use of atropine makes little or no impression on the pupil, it is often advisable not to go on so freely with the atropine, as the dragging thus produced rather tends to keep up than to allay the inflammation. The pain is best relieved by the frequent use of hot fomentations. Leeching, either with the artificial leech (Heurteloup), or with natural leeches (three or four to the temple), sometimes has a good effect on the pain, though it has little influence on the course of the inflammation. In many cases opiates give very decided and prolonged relief. Cocaine locally three or four times a day, or oftener, may be tried in some of the chronic forms, when atropine, on account of the abundant synechiæ, is contra-indicated. It appears sometimes to check the inflammation, though why this should be it is difficult to understand. Patients with iritis should, as a rule, have the temperature of their rooms kept as equable as possible, as they are often liable to relapses from exposure to cold.

The *treatment* sometimes required for cases of old iritis is iridectomy. This operation is called for in two different classes of cases. In the first place, to improve vision where the pupil is small, and to a great extent occluded, though the condition has not led to serious nutritive changes; and in the second place, with the object as well of preventing the complete destruction of vision which either the existing condition or the danger of recurrence of the inflammation has threatened. Iridectomy, when performed for iritis, should as much as possible be done at a time when all symptoms of inflammation have subsided, and the portion of iris excised should be large, so that in the event of a subsequent attack taking place, the coloboma, or artificial pupil, may be less likely to close up. At the same time, a large iridectomy gives the best chance for the re-establishment of the normal flow of aqueous fluid from the site of its secretion to that by which it leaves the eye, the interruption in which process may have, as already explained, such serious consequences. Iridectomy is urgently called for, and often followed by the most brilliant results, while the condition known as "iris bombé" is present, and the intraocular tension increased. The effect of iridectomy in this condition was one of the greatest of the many discoveries which ophthalmic surgery owes to von Graefe, and one which in great measure led him to adopt the same treatment so successfully in glaucoma. Even in cases where the tension is below normal, if there be not already blindness, iridectomy may prove of service. In recurrent iritis, too, an iridectomy performed at a time when the inflammation has subsided, often, though certainly by no means invariably, prevents the recurrence or lessens the frequency and severity of subsequent attacks.

Other operations which are performed for old iritis, such as attempts at separating the synechiæ, and sclerotomy, are either less safe or less efficient than iridectomy, and on that account not to be recommended. In the worst cases, where the iris has become friable or "rotten," and more or less atrophied from repeated inflammation, and where the attachments to the capsule are very extensive owing to the association with cyclitis, it is sometimes possible to improve vision and prevent further destruction taking place by removing the lens. An operation of this nature is most frequently called for in cases of severe sympathetic iritis.

When iritis is not set up by injury, the two most common causes are rheumatism and syphilis. It is difficult to determine in what proportion these two predisposing causes are accountable for the cases met with, as not only is there probably a very considerable difference in this proportion in different places, but it is by no means easy always to exclude the possibility of a syphilitic origin in any particular case. We may say with tolerable certainly, however, that syphilis is seldom responsible for less than ten per cent, or more than fifty per cent. of all the cases, so that on the whole the rheumatic form is decidedly the most common.

Rheumatic Iritis.—This form of iritis occurs most frequently during the prime of life, between the ages of twenty and fifty. There is often a distinct history of former rheumatic affections elsewhere, or there may be other parts affected with rheumatic inflammation at the same time. At other times there is no other rheumatic manifestation, and yet a great tendency shown to iritis from exposure to cold. Most frequently only one eye is affected, but both may be, either at the same time or within a short interval. The inflammation occurs with very varying degrees of severity, and the prognosis, even in the most severe cases, is favourable if proper treatment be adopted from the first, before extensive synechiæ have formed or cyclitis has become developed.

The *treatment* consists in avoiding changes of temperature or light and the use of the eyes, also in keeping up the dilatation of the pupil—in attention, in fact, to the general line of treatment for iritis. Besides this, great benefit is usually got from the use of salicin or salicylate of soda. Perhaps the best way of using salicin is for the patient to begin taking seven to eight grains every hour, remaining in bed all the time, and afterwards to take from ten to fifteen grains thrice daily for some time. In this way it is sometimes possible to cut short the attacks, and in almost all cases the pain is lessened. In obstinate recurrent cases it is well to avoid too much care in preventing draughts, &c., as the susceptibility of the patient may thereby be considerably increased. It is better to make some systematic attempts at a gradual hardening, and allow more and more exposure to the conditions which appear to bring about the attacks. At the same time two or three visits to Wiesbaden often do good. The baths, not the waters, are recommended by Pagenstecher, and these should not be begun until a few weeks at any rate have elapsed since the last attack of inflammation. In the distinctly rheumatic, other baths, such as Harrogate, Droitwich, Nauheim, Wildbad, Acqui, &c., may be recommended.

GONORRHŒAL IRITIS.—Closely allied to rheumatic iritis is the rare form which is undoubtedly associated with gonorrhœa. The inflammation is always severe, and the cause, owing to co-existing inflammatory changes in the joints is apt to be overlooked. Gonorrhœal iritis always seems to occur in both eyes, though not always quite simultaneously, and with, it may be, different degrees of severity in the two eyes. Successive attacks of gonorrhœa are sometimes accompanied each time by iritis, and in other cases, although no fresh inoculation takes place, a return of the joint affections may be accompanied by a recurrence of the iritis as well.

The local *treatment* is the same as for other forms of iritis. Iodide of potassium in large doses seems to be the best general treatment at first, followed, as the inflammation subsides, by quinine and iron.

SYPHILITIC IRITIS.—Iritis occurs as a secondary manifestation of acquired syphilis, and also in inherited syphilis. Though syphilitic iritis is very commonly met with in eye hospitals, it is by no means a common secondary symptom. The statistics of syphilologists differ widely in respect to its frequency—from one per cent. (Siegmund) to five per cent. (Bock). Hebra and Fournier each found three to four per cent. Iritis is for the most part a late secondary manifestation, making its appearance as the others are fading away, or after they have altogether disappeared. Sometimes, however, it runs its course contemporaneously with other secondary symptoms, and may even be the first to appear.

The hereditary cases of iritis mostly occur at about the time of puberty, and in association with interstitial keratitis. Some few cases are met with in early infancy, or still more rarely in intra-uterine life.

It is as a rule difficult, or impossible, to detect from the mere inspection of the eye alone when the iritis is due to syphilis. One has therefore generally to be guided by the presence or absence of other secondary symptoms in arriving at a diagnosis, or by the history in cases where the iritis exists alone. It should be remembered, too, that it does not by any means follow that iritis occurring in an individual who has at one time contracted syphilis is essentially specific in its nature although the presumption is in favour of such being the case.

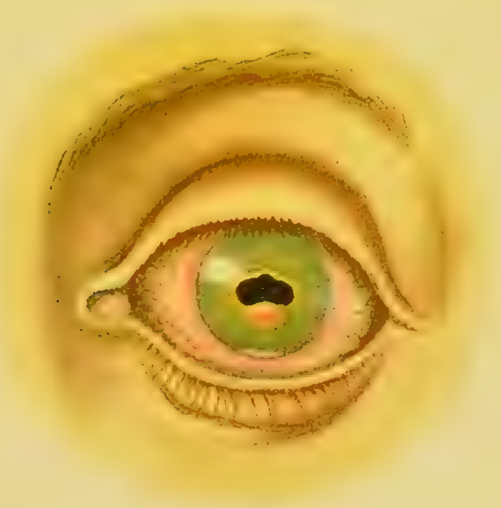

Fig. 70.—Gummatous iritis.

In some cases (about two per cent. of all, according to the average of statistics by a number of observers) the inflammatory changes in the iris, which are usually of a plastic nature, bear strong evidence of their syphilitic origin. Thus the development of more or less prominent yellowish vascularised nodules in the iris, or of localised tumefactions of the iris tissue, a condition known as *gummatous iritis*—(see Fig. 70)—is almost invariably due to syphilis. The gummata grow slowly, often causing very little pain or other evidence of inflammation, until they have attained some size. Their vascularity increases as they grow, so that while at first they appear as yellowish spots, they later on change their colour to a dirty brown. It is this alteration in colour which in doubtful cases mainly distinguishes them from tubercles. The latter, too, are as a rule more numerous, and mostly spring from the peripheral part of the iris in the region of the angle of the anterior chamber, whereas the gummata show a greater preference to develop in the parenchyma surrounding the pupil. The gummata may undergo fatty degeneration and become absorbed, leaving no trace, or only a slightly depressed atrophic spot at the sites where they existed. In other cases they increase in size, growing into the anterior chamber, and lead, with or without perforation of the cornea, to shrinking of the eye,—*phthisis bulbi*. The latter result is more uncommon than the former, and most likely to occur where the gummata originate in the peripheral portions of the iris, when they are frequently associated with the formation of similar nodules in the ciliary body.

Gummatous iritis does not, as the name might imply, belong as a rule to the tertiary period of syphilis, but to the secondary, although on the whole appearing rather later than the more common form.

Syphilitic iritis may occur in one or both eyes. No cause can, as a rule, be given for its occurrence—there has been no trauma, no exposure to cold, &c.

While the invariably bilateral character of gonorrhœal iritis seems to admit of the supposition that the inflammation is set up through the agency of some irritant product which is contained in the blood itself, the connection between the altered state of the blood in syphilis and syphilitic iritis is not so evident, on account of its frequently occurring on one side alone. It is of course possible that in some, perhaps in many cases, there may be a special vulnerability of the iris, so that slight and often unnoticed temperature changes may be the immediate means of setting up the iritis. Leber's view that the inflammation is determined by the accidental entanglement in the capillaries or smaller vessels of the iris, of minute coagula containing the specific virus, affords a better explanation both of the comparative infrequency of iritis in syphilis, and of its frequent unilateral nature when it does occur. No anatomical proof has yet, however, been given of its correctness.

The local *treatment* for syphilitic iritis should be carried out on the lines already laid down, with reference to the treatment of iritis in general. When the case is one occurring during the secondary period, and therefore undoubtedly specific, the patient should be kept on a simple but nourishing diet, and abstain altogether from alcohol and tobacco. The bowels should be regularly and quietly moved, preferably by the use of a saline aperient water, and mercurial treatment, except in the case of inherited syphilis, be begun at once and continued as long as possible.

There are, of course, different methods of using mercury, but treatment by inunction is, in my opinion, preferable to either internal administration or subcutaneous injection. If care be taken to keep the mouth clean by the frequent use of solutions of permanganate or chlorate of potash and a toothbrush, the inunction may be continued for weeks without inducing symptoms of mercurialism, which it should be remembered are accidents to be avoided, and not to be looked upon as indicating that sufficient mercury has been taken.

Whatever view may be held as to the ultimate relative advantage of the mercurial or non-mercurial treatment of syphilis, the eye is too important an organ for us not to take advantage, when treating the syphilitic changes which occur in it, of the long-established fact of the value of mercury in causing the disappearance of secondary manifestations.

Cases of plastic iritis, that is to say, as distinguished from the gummatous variety, which begin six months or more after the disappearance of secondary symptoms, should, on the other hand, not be treated with mercury, but in the way recommended in cases of rheumatic iritis, or with iodide of potassium. When the patient has completely recovered from his iritis, iron in some form may advantageously be prescribed, or better still, if his circumstances admit of it, he may be sent to some chalybeate bath.

The forms of iritis already described, along with sympathetic iritis, which is discussed in the chapter on Sympathetic Ophthalmitis, are the main forms of the plastic variety.

SEROUS IRITIS.—This variety of iritis, which receives its name on account of the exudation being on the whole more serous than fibrinous, is almost invariably associated with a cyclitis or choroiditis. It is much more frequent in women than in men. According to Horner the proportion in the two sexes is as ten to three. Corresponding to the comparative absence of a plastic exudation, serous iritis is a less painful, more insidious, and usually more chronic affection, than most of the forms of plastic iritis. The irritation it produces is indeed often so slight, that the attention of the patient is first called to the eye by the certain degree of haziness of vision to which it gives rise. The back of the cornea is then found on examination to be covered, mainly in its lower quadrant, by a number of minute brownish specks, while the aqueous humour is at the same time more or less turbid. The circumcorneal injection is usually slight and most marked at the lower part, with a great tendency, however, to become more apparent if the eye be kept open for some time, or if it be rubbed.

The small deposits, which lie on the membrane of Descemet, consist mainly of pigment, along with leucocytes and fibrin. This condition often receives the name of keratitis punctata, although it is not, properly speaking, a keratitis. Descemetitis is another term applied to the same appearance (see Fig. 71). The pigment evidently comes from cells, and is not altered blood pigment. The larger spots are due sometimes, it would appear, to proliferation as well of the underlying cells of Descemet's membrane.

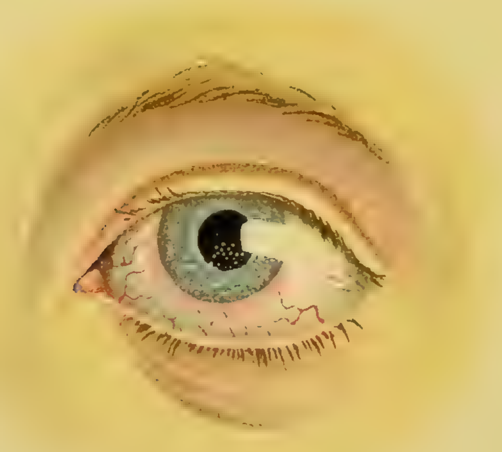

Fig. 71.—Serous iritis, showing the condition known as keratitis punctata, or Descemetitis.

In the first stage of the inflammation there are no synechiæ, or only very trifling ones. Often a very distinct hyperæmia of the papilla is to be made out with the ophthalmoscope. In the majority of cases synechiæ begin to form after the inflammation has remained for some weeks in the first stage. At the same time the hyperæmia of the papilla becomes more marked, and diffuse white floating opacities make their appearance in the vitreous. These changes are accompanied by alterations in the intraocular tension, which is first increased, and afterwards very often considerably diminished.

The *prognosis* in this form of iritis depends much on the general health of the patient, as well as of course on the extent to which the uveal tract as a whole has participated, and on the completeness of the synechiæ. A number of cases end in complete recovery, but more frequently the recovery is incomplete. In some cases only slight changes in the vitreous occur at all, so that recovery takes place after the inflammation has remained for some time in the first stage, the symptoms being mainly confined to the anterior portions of the eye. In others, again, the more or less dense vitreous opacities clear away after longer or shorter periods, and no further harm results, if the formation of synechiæ has been obviated by keeping the pupil dilated. Owing, however, no doubt to the alterations which the uveal inflammation brings about in the nutrition of the eye, there is a tendency, in cases where opacities of the vitreous have been marked and persistent, towards the gradual development of cataract, which almost invariably then begins with an opacity confined to the posterior portion of the lens,—what is called *posterior polar cataract.* The worst cases are those in which extensive synechiæ have formed, with occlusion of the pupil, and the termination, as in other forms of iritis, either in secondary glaucoma, or detachment of the retina, with shrinking of the eye.

The *etiology* of serous iritis, or more correctly serous iridocyclitis, is not always very clear. The preponderance of cases in women, already referred to, shows that syphilis cannot be at all events a general cause. Probably Horner's view is the correct one, that it results from some pathological condition of the blood or blood-vessels. He points out, for instance, that it is this variety of iritis which is apt to occur after fevers or severe illnesses. Often there are menstrual disturbances, or actual disorders of the sexual organs, in women affected with this disease; all are more or less anæmic or chlorotic. When it occurs in men, it is for the most part, too, in such as are ill-nourished and anæmic.

In the *treatment* greater care must be taken than in the case of the plastic variety of iritis not to use atropine too freely, on account of the tendency there is towards increased tension. The pupil should at first be kept dilated, or semi-dilated, with as weak a mydriatic as possible. When the deposits at the back of Descemet's membrane are numerous and large, it is well to perform paracentesis of the cornea, taking care not to allow the aqueous humour to escape too rapidly. The tapping of the anterior chamber may be frequently repeated if undertaken with proper antiseptic precautions. It appears to have a beneficial influence on the inflammation, and to favour the absorption and disappearance of the exudation into the vitreous as well. When these vitreous opacities are dense, wet packing and subcutaneous injections of pilocarpine are often of use in promoting absorption. Where there is marked increased tension, especially in elderly people, I have found sclerotomy performed with the triangular-shaped knife (keratome) of great use. Owing to the prevalence of anæmia, iron in some form or other, and arsenic, are of use. Change of air, with regular moderate exercise, should also be recommended.

TRAUMATIC IRITIS.—*Purulent Iritis.*—Iritis may be set up by direct injury to the iris, or by injury to the contiguous parts of the eye, the cornea, lens, or ciliary body. The injury may be an accidental one, or be caused by some operation, such as the extraction or discission of a cataract. The severity of the inflammation depends greatly upon whether or not septic matter or any foreign body has remained in the eye. These almost invariably give rise to a purulent inflammation. The pus which is then excreted from the inflamed iris not only infiltrates its tissues, collecting mainly between the endothelial covering and the stroma, but also falls to the bottom of the anterior chamber as hypopyon. At the same time some of the vessels give way, causing hæmorrhage into the iris. Purulent iritis may, too, form only a part of a general suppurative destruction of the eye, the result either of external local changes, or of septic emboli in the choroid. Sometimes a traumatic iritis does not occur at all, or only slightly, at the time of an injury, but is set up at some subsequent period by changes which occur in the eye which lead to persistent dragging on, or irritation of, the iris.

The *treatment* of traumatic iritis will depend greatly upon the nature of the injury as well as upon the severity of the inflammation. If the lens has been wounded, or if a foreign body be lodged in the iris or in the anterior or posterior aqueous chamber, some operative interference will usually be called for. Where there is no foreign body, but where the nature of the accident and the presence of much hypopyon render the septic character of the inflammation undoubted, it may often be advisable to make a small opening into the anterior chamber and wash out the contents with a weak antiseptic. This proceeding should not be too rashly undertaken, as many cases are best left alone.

TUBERCULAR IRITIS.—This is a rare form of iritis, which may occur in one or both eyes, and most commonly makes its appearance before the period of full growth. Horner found the greatest frequency after the fifth year and before puberty. The inflammation begins as an ordinary serous or plastic iritis and is often preceded by ill-health and loss of flesh, and accompanied by, sometimes very considerable, swelling of the surrounding lymphatic glands. Tubercles in the shape of small yellowish nodules, usually in considerable numbers, make their appearance, and the iritis shows a tendency to become chronic. The tubercles may become confluent, or disappear and be replaced from time to time by others. Tubercular disease may or may not exist in other organs at the same time.

Some cases completely recover, others again lead to shrinking of the eye or to perforation and protrusion of a tubercular mass externally. There is seldom any difficulty in the *diagnosis*. Gummata of the iris, which they somewhat resemble, are more vascularised, and occur besides at a different time of life. The worst cases are those in which the tubercles become confluent, and in such there is not only greater danger for the eye, but the prognosis is unfavourable with respect to the life of the patient as well. These cases were long known under the name of *granuloma* of the iris before their true tubercular nature was established. Jacobs of Dublin considered them to be tubercular, and described them as cases of tubercular iritis, but it is only comparatively recently that his views have been confirmed by inoculation experiments on animals. Horner seems to have considered some, at all events, of the cases in which the nodules in the iris are multiple, and exhibit little or no tendency to become confluent—for which, too, the prognosis is much more favourable—cases of lymphomatous iritis. It seems probable, however, that they are all tubercular. Tubercular changes in the iris are thus a much earlier manifestation of the diathesis than tubercle of the choroid.

In the *treatment* the possibility of the disease in the iris acting as a source for self-infection must always be kept in view. This danger is apparently greatest in the cases of granuloma which lead to perforation, but it is also present in the form which ends in phthisis of the globe. There can be no doubt, therefore, that the proper treatment in the worst cases is enucleation. In some cases an isolated tubercular mass may be excised (along with the portion of iris in which it is situated), but this treatment is only rarely successful. The iritis must be treated on the ordinary lines, and the patient's strength

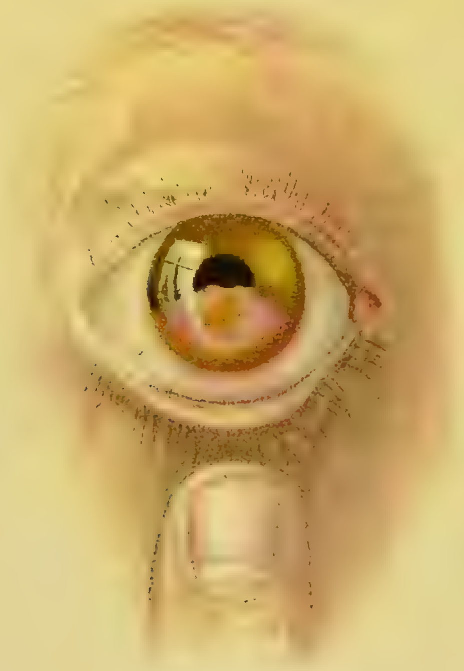

Fig. 72.—Tubercular iritis.

kept up by nourishing food, and the exhibition of anti-strumous remedies.

Other varieties of iritis are described, such as scrofulous iritis, and those following different fevers. These do not exhibit any special features indicative of the particular illness with which they are associated. They for the most part assume the serous variety, or they are metastatic, resulting from embolism of the vessels of the iris, and are then associated with a similar inflammation of the choroid.

Iritis may also come on in connection with neuralgia of the fifth nerve, and appears especially prone to do so where the neuralgia is due to some trauma. The prognosis in these cases is on the whole bad.

CYCLITIS.

Inflammation of the ciliary body, or cyclitis, is best considered in this connection, as it is so frequently associated with iritis. One of the most common symptoms of cyclitis, and that which is, from a diagnostic point of view, perhaps of the greatest importance, is tenderness to pressure over the ciliary region. This tenderness, which is very different in degree in different cases, is often more marked at certain spots than at others. When this is the case there can be little doubt that inflammation of the ciliary body exists, unless the iritis be very severe.

The character of the exudation from the inflamed ciliary body may, as in the case of iritis, be mainly serous or plastic or purulent, and the symptoms may differ more or less accordingly. The exudation passes from the two free surfaces of the ciliary body into the posterior aqueous chamber and into the vitreous chamber.

A constant symptom of cyclitis is therefore more or less opacity of the vitreous. Often this can only be inferred from the defect of vision which it produces, as, owing to the presence of synechiæ, and the difficulty of obtaining dilatation of the pupil, the objective determination of the opacities may be a matter of difficulty. The exudation from the anterior portion of the ciliary body leads to synechiæ, precipitations on Descemet's membrane, obliteration of the pupil, and in bad cases to an agglutination between the posterior surface of the iris and the capsule of the lens. In the case of purulent cyclitis there is also hypopyon. That portion of the exudation of plastic or purulent matter which passes through the ciliary processes into the vitreous chamber may remain confined to the immediately surrounding regions, or permeate the vitreous more or less extensively, causing it to become more fluid. If absorption does not rapidly take place, changes occur in the exuded matter leading to the formation of filamentous or even membranous shreds of a low form of connective tissue. Where there has been an excessive exudation, the subsequent shrinking which takes place in this tissue may result in detachment of the retina, and consequent complete loss of vision. In many cases, where the exudation has accumulated, as there is a tendency for it to do, at the anterior portion of the vitreous, subsequent contraction, taking place during its organisation, leads to displacement of the lens. Most frequently this displacement is forward, so that the anterior chamber becomes more or less shallowed; sometimes, owing to the contraction being more in one direction than in others, some portion of the lens is tilted forward, and the chamber is shallowed in one direction, and deepened in the opposite.

In some old standing cases the anterior chamber is deepened, so that a funnel-shaped depression is observed to exist at its centre. This is produced by contraction in an antero-posterior direction, which brings about an approximation of the lens and the optic papilla, to which the organised exudation has formed an adhesion. In such cases there is at the same time some diminution in the intraocular tension.

Cyclitis may be set up traumatically by an injury from without, or by some alteration taking place within the eye. A severe blow on the eye may be sufficient, but more common causes are penetrating wounds, and the introduction of foreign bodies either into the ciliary body itself, or into some of the surrounding parts, as, for instance, the posterior aqueous chamber or the lens. In some cases, where foreign bodies have become encapsuled, they may, from some cause or other, become suddenly dislodged, and then set up cyclitis months or years after the accident which drove them into the eye. It may be that, having lodged in the lens, thus producing traumatic cataract, the gradual disintegration and absorption of the lenticular structure leads to an alteration in the position occupied by the foreign body. The new position is apt to be one which causes more irritation of the tissues of the eye and cyclitis.

Traumatic cataract alone, *i.e.*, without the presence of any foreign body, may in various ways set up cyclitis. This may happen if a rapid swelling takes place where the lesion in the capsule has been small, and the swollen lens matter does not readily make its escape into the aqueous chamber. Again, it may happen if the iris, owing to its having formed adhesions to the wound in the capsule, is subjected to more or less constant dragging. A dislocated lens is also very apt to set up cyclitis, more especially if it has at the same time undergone advanced degenerative changes.

While irregular and septic wounds in the ciliary region almost invariably lead to an immediate and usually purulent cyclitis, very little irritation may result from clean and aseptic wounds in the same situation.

The *treatment* in the case of foreign bodies lodged in the eye is discussed in Chapter IX. Wounds of the ciliary region should be carefully washed with some antiseptic lotion, and any separation of the lips as much as possible prevented by cutting off or replacing the intervening structures. Swollen lens matter, when causing irritation, must be removed in the manner described in the chapter on Operations. A dislocated lens must be extracted, and where any dragging on the iris has set up irritation, or appears likely to do so, it will be necessary to perform iridectomy in such a position as to free if possible the portion dragged on.

When cyclitis is once set up, the treatment should consist in keeping the pupil dilated with atropine, in applying hot fomentations to the eyes, and in leeching over the temples. The eyes should be shaded, and the patient not allowed to read, or use them for any work near at hand. In purulent cases it is well to apply a pretty tight bandage over the affected eye, as this seems to aid in checking the tendency to panophthalmitis—that is, to an extension of the purulent inflammation to the choroid.

TUMOURS OF THE IRIS.—Both simple and malignant tumours are met with in the iris. They are rare, and not of particular interest. Two kinds of cyst occur—the *epidermoid* and the *serous.* The former only appears to come after there has been some penetrating wound of the cornea, and the latter is usually also of traumatic origin. In fourteen cases of cysts of the iris observed by Snell, ten had been preceded by trauma. Hulke again found out of nineteen fifteen in which there had been some injury, and Rothmund was able to trace a traumatic origin thirty-one times out of thirty-seven collected cases. The proportion of traumatic cases is therefore from these statistics about four in five, and may in reality not improbably be greater. At the time of the accident some small portion of skin or corneal epithelium, or a piece of an eyelash, is driven into the anterior chamber, and there proliferates, assuming usually a cystoid type of growth. Experiments conducted by Hosch have shown that these cysts are most likely to grow when along with the epidermis some glandular tissue is transplanted to the eye. According to Rothmund, who first proposed the division into epidermoid and serous cysts, the time elapsing between the accident and the formation of an epidermoid cyst is at least two months, and may amount to several years. The serous cyst seems mostly to extend from the margin of the anterior chamber. It is, in fact, a kind of cystoid degeneration of the iris, leading to the formation of a diverticulum at the angle of the chamber. The prognosis is bad in both these forms of cyst, as they are apt to go on growing, and eventually lead to destruction of the eye.

The *treatment* consists in excising them as soon as possible, along with the portion of iris to which they are attached. Care should be taken not to rupture the cyst wall during removal.

Like other parts of the eye, the anterior chamber may be visited by a *cysticercus.* In the cases which have been observed, and which have occurred almost exclusively in Germany, the surrounding cyst has sometimes been clear, at other times purulent.

Sarcoma of the Iris is usually an extension from the ciliary body, which is a common site for malignant tumours within the eye. It may be pigmented (melanotic) or not. Only in the latter case is there any possibility of mistaking it for a gummatous or tubercular mass. Primary sarcoma of the iris does, however, occur. Cases have been successfully operated on by Little and others, by removal of the iris in which the tumour grew. Their early diagnosis is therefore a matter of importance.

Proliferation of the pigmentary tissue of the iris, or *melanomata* of the iris, are met with as dark pigment spots or small growths. They usually remain stationary, and do not interfere with vision, but in some cases they appear to have been the starting-points of sarcomatous growths.

Lipomata, *Angiomata*, and other varieties of tumours, are only of the utmost rarity. I have met with one case of nævus, not only of the iris, but of a persistent pupillary membrane at the same time.

Alterations in the Iris produced by Injury.—Lacerations of the iris may be produced by penetrating wounds. When the history of the accident points to the impaction of a small body, the appearance of the laceration of the iris is strongly suggestive of its presence somewhere in the eye. Sometimes the laceration has taken place at the pupillary margin,—more frequently, however, it is elsewhere; and the more peripheral it is, the more importance does it acquire from a diagnostic point of view, when the question arises as to whether or not a foreign body is lodged in the eye. With the more central wounds there may usually be made out the corresponding wound in the lens, while in the case of peripheral wounds of the iris, a trauma of the lens is neither of so frequent occurrence, nor so easy of observation when it has occurred. The laceration may cause bleeding, which is usually slight, and the hyphæma, or collection of blood in the anterior chamber, to which this gives rise, is as a rule rapidly absorbed. Synechiæ almost always occur in cases where the wound is situated not far from the pupil, unless the pupil has been well dilated soon after the accident.

A simple wound of the iris is not likely to cause iritis, but when, as so often happens, the lens is wounded at the same time, or a foreign body is lodged in the eye, the chances of inflammation being set up in the iris are very great. The *treatment*, too, will vary according to the severity of the accident. When the iris alone is injured, a few days' dilatation of the pupil with a mydriatic and rest to the eyes is all that is usually required. Complications must be treated as they arise, after the manner explained in connection with traumatic iritis and cataract, and in the chapter on foreign bodies in the eye.

Various *injuries to the iris* are produced by blows on the eye; the simplest of these, though not the most frequent, is a rupture of the sphincter muscle. This accident is in my experience not so rare as the paucity of literature on the subject might lead one to suppose. Of itself it is of little importance, but it may be associated with other changes liable to be produced by contusions. Sometimes a rupture takes place in the direction of the radial fibres of the iris, but without involving the sphincter. This, too, may occur spontaneously in cases where the iris is atrophic, and dragged on in some particular direction.

A more common accident, produced by severe contusions, and often associated with rupture of the choroid, is a separation of more or less of the iris from its peripheral or ciliary attachment. This accident (see Fig. 73), to which the name of *irido-dialysis* is given, is always accompanied at the time by hyphæma. If other parts of the eye are uninjured, it may occasion monocular diplopia, if the eye, from an error of refraction or owing to the state of accommodation, is not focussed for the object looked at. Some dazzling is also produced, owing to the irregular refraction through the peripheral portions of the dioptric media of the eye. The separation causes a flattening of the corresponding portion of the pupil.

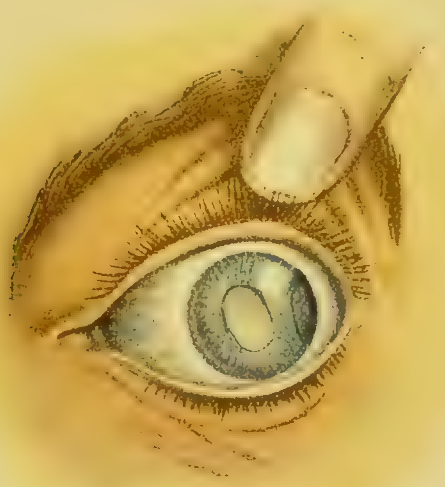

Fig. 73.—Irido-dialysis and cataract.

All degrees of irido-dialysis are met with. When very slight, it can only be easily diagnosed by throwing light into the eye with the ophthalmoscope, when the red reflection from the fundus can be seen at the periphery of the iris, as well as through the pupil. A spontaneous re-attachment of the iris to the ciliary body probably never occurs. A case is recorded from the Dublin Eye Hospital, in which, under atropine, and a bandage applied very shortly after the accident, complete cure took place. This fortunate result is, however, so rare, that it could hardly be expected to occur in any particular case. Occasionally, in performing iridectomy, if the iris be too forcibly drawn upon by the forceps, a separation occurs at its periphery.

Another and very curious result of blows on the eye is the complete or partial *retroversion* of the iris. This gives rise to the appearance of a complete absence of any iris at the position of the lesion. When partial, it is usually possible to see the folding of the iris, where the inverted portion joins on either side the portions which remain *in situ.* The existence of the folded back portion can also be inferred from the fact that the ciliary processes are not visible with the ophthalmoscope, notwithstanding that no iris intervenes between the cornea and the margin of the lens.

Temporary or permanent *dilatation of the pupil* may also result from a blow on the eye. Often the dilatation is irregular.

Where the lens is dislocated or absent, and thus the natural support which the iris gets by its apposition to the capsule in the normal state is interfered with, or altogether lost, the iris is observed to shake with the movements of the eye. This tremulous condition of the eye is called *irido-donesis.* It should direct attention to the lens, an abnormality in which is the most common cause, though sometimes a not inconsiderable degree of shaking may occur in cases where, from some cause or other, the posterior aqueous chamber is unusually deep.

In old cases of iritis, especially if complicated by any condition which gives rise to constant dragging on the iris, *atrophy* of the iris takes place. The tissues may be so thin as to admit of more or less light passing through from the fundus on ophthalmoscopic examination; often there are actual rents here and there. Atrophy of the iris is common in the last or degenerative stages of glaucoma.

CONGENITAL ANOMALIES OF THE IRIS.—*Coloboma of the Iris.*—(see Fig. 74)—is a congenital defect, which is due to non-closure of the fœtal fissure. It may or may not be associated with a similar congenital defect in the choroid. The coloboma may be of all sizes, up to one-fourth of the whole iris. It always occurs downwards, or downwards and inwards, and generally, though often not to the same extent, in both eyes. It may occur in several members of the same family, but there does not appear to be the same tendency to this as in many other congenital defects. The coloboma may be either total or partial—that is to say, it may extend right up to the ciliary body, or close before reaching so far back. In some rare instances closure has been found to have taken place at an intermediate point, so that the coloboma is bridged.

Closely allied often to coloboma of the iris is the condition known as *correctopia*, or eccentric displacement of the pupil. Owing to faulty development of the muscles of some portion of the iris and ciliary body, the pupil becomes drawn up, from the preponderance of action of the more complete portions. Sometimes the muscular defect is due to other causes than coloboma, and may then be either congenital or accidental.

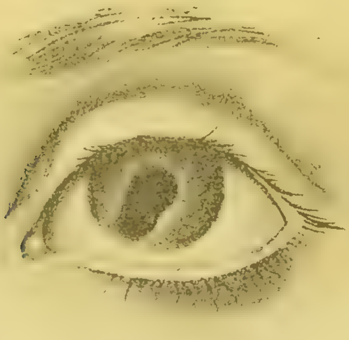

FIG. 74.—Congenital coloboma of the iris (downwards and inwards).

Aniridia.—Not so common as coloboma, but still not of very rare occurrence, is a congenital absence of the iris—*aniridia*, or *iriderœmia*, as it is called. This condition is most commonly met with in several members of the same family, and is inherited. It almost invariably occurs in both eyes at the same time. The nature of the arrest in development, which gives rise to aniridia, is apparently not understood. The appearance which the total absence of the irides gives to the eyes is peculiar, and not altogether like that which most resembles it, viz., maximal dilatation of the pupils. In all cases which have come under my own observation there has been at the same time a ring of pigmentation in, or immediately behind, the peripheral portion of the cornea. In many cases there is some defect in the lenticular system,—either congenital displacement of the lens, due to some faulty development of the suspensory ligament, or more or less opacity of the lens, usually stationary. Myopia, or defective accommodation are frequent concomitants, and there is usually as well more or less defect of sight. Patients with this defect are bothered by dazzling, and acquire the habit of screwing up their eyes. There is often at the same time photophobia, and, in the cases where the associated defects are most marked, nystagmus.

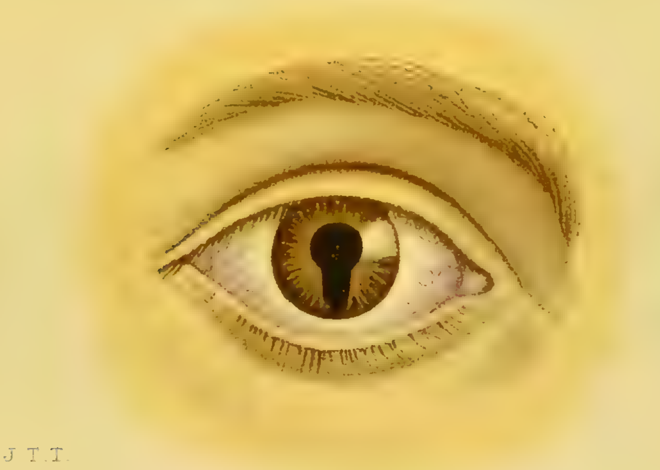

FIG. 75.—Congenital coloboma of the iris (downwards).

Albinism.—The general absence or defective development of pigment throughout the body, which characterises the albino, gives to the iris a very peculiar appearance. It has usually a pinkish or faint lavender colour, and the details of its structure are more evident than in an iris in which there is a normal amount of pigment present. When the eye is examined with the ophthalmoscope, a considerable amount of light, reflected from the fundus, is found to pass through the iris, and it is this translucency, as well no doubt as the associated absence of pigment in the choroid, which gives rise to the dazzling from which albinos suffer, and which causes them constantly to screw up their eyes. The vision is more or less imperfect, and there is some degree of nystagmus in all cases. Some cases are met with where the pigment is not altogether absent. In these there seems as a rule to have been a gradual development of pigment since birth, with a corresponding improvement of vision. Albinism is sometimes met with in several members of the same family.

Little can, as a rule, be done to improve the sight. Some find comfort, without any visual improvement, by the use of darkened glasses. Stenopaic apertures are only useful in cases where the nystagmus is slight. Any operative treatment, such as tatooing or creating a nebula in the cornea, is hardly justifiable.

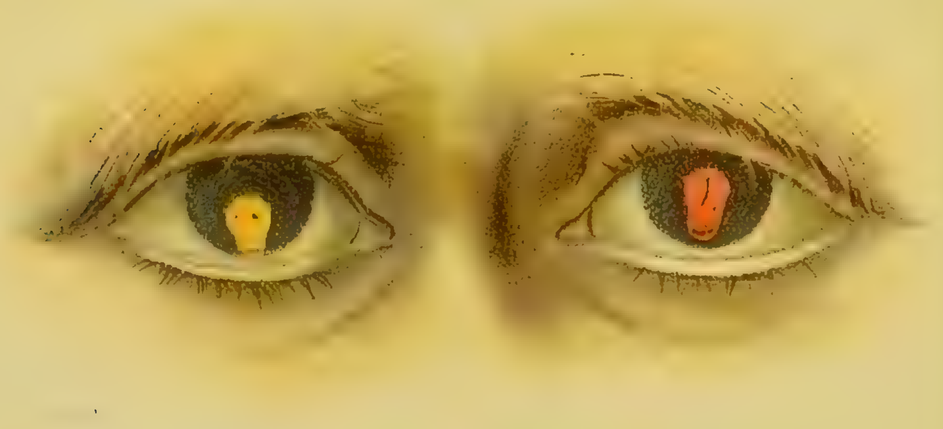

FIG. 76.—Double coloboma of the iris with correctopia (right eye).

The iris of the new-born infant is grey or blue in colour and either remains so, or gradually accumulates pigment, so as eventually to become more or less brown. These changes take place as a rule very early in life, but are sometimes deferred till the third or fourth year, or even later.

The influence of heredity on the colour of the eyes is very marked, and there seems no greater tendency to inherit from either parent. It has been found, for instance, that of the offspring of parents with differently coloured irides—that is, the irides of the one parent dark and of the other light—fifty per cent. have dark, and fifty per cent. have light irides; while when both parents have light or dark irides, ninety-six per cent. of the offspring present the same colour as the parents, and only four per cent. not. Of these four per cent., most have both eyes the same, inheriting probably from some remote ancestor; while a few present the appearance known as *heterochromia*, where the one iris is brown and the other blue

Another form of heterochromia is met with,—that in which part of the iris is of one colour and part of another. This, as in the more normal cases of iris-coloration is usually developed very early in infantile life, but may also make its appearance in late childhood. Fig. 77 represents a case in which the condition was tolerably symmetrical on the two sides.

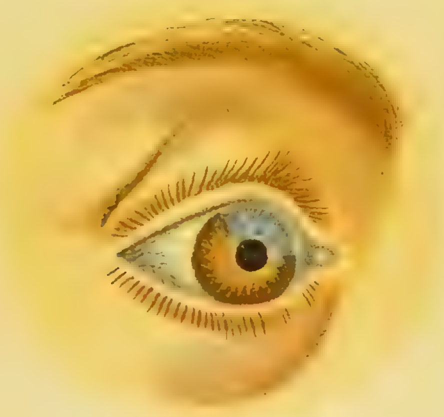

FIG. 77.—Heterochromia iridis.

In normal eyes the pupillary membrane, which, during the greater part of intra-uterine life, stretches across the pupil, only remains persistent in that part which covers the iris, whose endothelial layer it becomes. It is not a very uncommon thing to find, however, small portions of the membrane stretching across the pupil in the shape of one or more fine threads. To this condition the term *persistent pupillary membrane* is applied. Occasionally a considerable portion of the membrane persists, and is adherent to the capsule of the lens, usually at the same time having filamentous attachments to the iris. This membranous form produces considerable defect of vision, while the more common filamentous form is of no importance whatever in this respect. Without careful examination the fine threads might easily be taken for fibrinous remains of an old iritis, but they can be distinguished from these by observing that they spring from some portion of the anterior surface of the iris, usually by two or more fibrous roots, while an iritic exudation comes from the lower surface of the iris, or at most adheres to the pupillary margin. Only the rare membranous form calls for any operative interference. I have seen several cases in which discission of the lens was necessary, on account of the amount of amblyopia which the condition occasioned.

Foreign Bodies in the iris are rare, as the force which impels them so far is usually sufficient to carry them into the posterior aqueous chamber, lens, or vitreous. Their *diagnosis* and *treatment* is considered in Chapter IX.

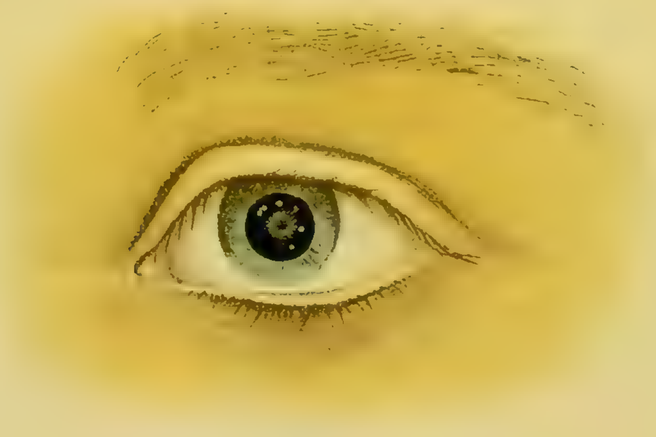

Fig. 78.—Capsulo-pupillary membrane with white spots on capsule.

Anterior Chamber.

Individual differences exist in healthy eyes in the depth and shape of the anterior chamber. Changes are also continually taking place in health, according to the state of accommodation. When the eye is accommodated for near objects, the middle of the chamber is shallower, and the periphery deeper, than in the case of accommodation for distance. This is owing to the increased thickness of the lens, which mainly arises from the greater curvature of its anterior surface, and to the retraction of the periphery of the iris, by the contracted ciliary muscle, of the accommodated eye. In very young infants and very old people the chamber is shallower than at other periods of life. In the former case this is owing to the globular shape of the lens and the imperfect development of the eye altogether; in the latter, to increase in the size, and possibly also to advancement, of the whole lens.

The anterior chamber is pathologically increased in depth in two ways; by an alteration in the normal position of the lens, and by a change in the normal curvature of the cornea. Thus we often find a deep chamber when the lens is absent or dislocated, and also when a hypersecretion, as in the case of serous iritis, causes it to be pushed backwards; or, again, in old-standing cases of irido-choroiditis or cyclitis, when a retraction takes place, owing to the shortening of organised exudation in the vitreous chamber. It is abnormally deep, too, in cases of conical cornea, cornea globosa, and staphyloma of the cornea.

Diminution in depth of the anterior chamber results from causes which lead to advancement of the iris or lens, or flattening of the cornea. Exclusion and occlusion of the pupil give rise to an accumulation of aqueous humour behind the iris, which is consequently pushed forward, and the chamber in that way shallowed. Shallowing of the chamber is also met with in glaucoma, and in the second stage of intraocular tumours. In old cases of irido-cyclitis, too, and in some cases of detached retina, the anterior chamber is shallowed, owing to atrophy of the vessels of the iris and ciliary body, and a consequent diminution in the secretion of aqueous humour.

Alteration of the *shape* of the chamber, retraction of the periphery, and advancement of the pupillary portion of the iris, is met with, and is an important point in the diagnosis of metastatic choroiditis in children, or what has been called *pseudo-glioma*.

The *contents* of the chamber, too, are subject to pathological alterations. Thus the normal clearness of the aqueous is disturbed in many cases of iritis and in glaucoma, and dislocation of the lens may take place into the chamber. Along with septic inflammation of the cornea, or purulent iritis, or cyclitis, pus may collect in the chamber (hypopyon). Injuries to the vessels of the ciliary body, or iris, may lead to a collection of blood taking place in the chamber (hyphæma). This condition of hyphæma may occur spontaneously after cataract extraction, in irido-cyclitis, in hæmorrhagic glaucoma, or intraocular tumour. It has also been met with rarely as a kind of vicarious menstruation, or as a manifestation of the hæmorrhagic diathesis. Finally, foreign bodies may lie in the anterior chamber, and may or may not be surrounded by purulent exudation, according to their nature and the time they have been in the eye.

CHAPTER VIII.

DISEASES OF THE CHOROID AND VITREOUS.

THE CHOROID.—A certain portion of the choroid, viz., that which lies anterior to the equator of the eye, is altogether hidden from view. Pathological changes affecting this portion can therefore only be inferred from the symptoms to which they give rise. The rest of the choroid is also more or less concealed by the pigment contained in the hexagonal cells of the retina. When this pigment is scanty, the larger vessels of the choroid, as well as the intervascular spaces, are visible on account of the transparency of the rest of the retina. The appearance presented by a normal choroid on ophthalmoscopic examination is described in Chapter I.

The blood supply to the choroid is mainly through the short posterior ciliary arteries, of which there are about twenty. These vessels enter in the vicinity of the optic nerve, at the back of the eye, and do not anastomose to any extent with each other. They form, however, pretty free anastomoses with the recurrent branches of the long anterior and posterior ciliary arteries. A considerable portion of the anterior part of the choroid is therefore supplied by these long arteries, and either by branches from the trunks themselves, or from the arterial rings which they form in the ciliary body.

The blood returns through veins, the arrangement of which is very different from that of the arteries. The venæ vorticosæ, from four to six trunks, collect all the blood from the choroid, as well as much which has supplied the anterior portions of the eye. The numerous choroidal veins mainly join these trunks, which pass out of the eye near its equator. Some open into anastomoses, which are formed here and there between the contiguous vortex veins behind the equator of the eye.

The choroid is only firmly adherent to the sclera behind at the entrance to the optic nerve, and anteriorly through the ciliary body. The attachment to the sclera in other situations is merely through the vessels which penetrate that membrane to reach the choroid. The pigment layer of the retina adheres closely to the choroid, so that when the retina becomes detached it leaves this layer behind.

Four distinct layers are recognised in the choroid proper. These are, from the hexagonal retinal pigment backwards—(1.) the vitreous membrane, or lamina vitrea; (2.) the layer of small vessels and capillaries—the chorio-capillaris; (3.) the layer of large vessels; and (4.) the membrana suprachoroidea. All these parts are held together by a stroma containing pigmented and unpigmented cells of different shapes. Some of the cells are unstriped muscular fibres, the function of which is not known.

The nerve supply of the choroid is through the ciliary nerves, which are branches of the third and fifth cranial nerves, and of the sympathetic. The direct or long nerves, two or three in number, are branches of the naso-ciliary. The short pass from the ciliary ganglion. Both long and short nerves pierce the sclera not far from the entrance of the optic nerve.

CHOROIDITIS.—As the extent to which the normal choroid is visible with the ophthalmoscope depends on the condition of the hexagonal pigment cells, so also does the visibility of pathological changes in the choroid. Considerable alterations may have taken place where there is yet nothing to be seen with the ophthalmoscope; indeed, in some instances, it is only when the pathological process in the choroid involves the retinal pigment cells that ophthalmoscopic changes are observed. Strictly speaking, then, in the case of inflammations of the choroid, characteristic ophthalmoscopic appearances are only seen when the retina is inflamed as well,—that is, when the condition is one not only of choroiditis but of *choroido-retinitis.* From a clinical point of view, however, an inflammation beginning in the choroid, though afterwards spreading to the outer portions of the retina, is a choroiditis. Such inflammations have altogether a different origin from cases of true retinitis, or inflammation of the nerve elements, or of the connective tissue of the retina.

Inflammation in the choroid may be serous, plastic, or purulent. Choroiditis, besides causing retinal changes, may give rise to more or less marked changes in the transparency and consistency of the vitreous. In two distinct clinical forms, too, the sclera is at the same time weakened, so that alterations in the shape of the eye take place, leading to more or less disastrous consequences.

There is a great tendency to a patchy arrangement in choroidal inflammation, or to what is called disseminated choroiditis, but in some cases, and more especially in the serous and purulent varieties, the inflammatory changes are diffused over the whole membrane.

It is difficult to arrive at any very satisfactory classification of cases of choroiditis, for the reason that it does not seem possible to tell with any degree of certainty to what pathological changes particular objective appearances, as revealed by the ophthalmoscope, are due. In fact it is certain that much the same appearances may be presented by very different processes. While an anatomical classification may be interesting from the point of view of scientific pathology, it is of little use in the practical or clinical sense. The following clinical types are, I think, sufficiently distinctive to justify their being separately considered:—(1.) Disseminated choroiditis, atrophic and exudative; (2.) Senile central choroiditis; (3.) Syphilitic choroiditis; (4.) Sclero-choroiditis, anterior and posterior; and (5.) Purulent choroiditis (traumatic, embolic, and metastatic).

Disseminated Choroiditis.—The ophthalmoscopic appearances met with in disseminated choroiditis are due to localised exudations into the choroid, alterations in the retinal pigment, and eventually also to atrophy of the choroidal stroma. The patches of exudation and degeneration, which sooner or later become visible, vary greatly in shape and size. Their average size may be perhaps roughly taken as about one-quarter of the disc in diameter. They are often approximately circular, but they may be of any other form, not unfrequently crescentic, or dumb-bell-shaped. By their confluence they assume all kinds of irregular forms. They differ in appearance according to the stage arrived at by the inflammation. Different foci of inflammation may exhibit different stages at the same time. In the beginning the spots are yellowish, or reddish yellow, and show little or no trace of choroidal vessels. Their margins are not very sharply defined, and there is occasionally some degree of prominence evident. These appearances correspond to the stage of hyperæmia and exudation. Old-standing patches, which have reached what may be called the degenerative stage, are white, with traces of the remains of choroidal vessels, and they are not prominent; their margins are sharply defined, even punched-out looking, and often bordered with pigment. The white appearance of the patches is due not only to the cicatricial changes which take place in the exudation, but to more or less reflection as well from the sclera, which is less obscured than when covered by normal choroid. As the transition from one stage to another takes place, the change to white is seen first to occur in the centre of the patches.

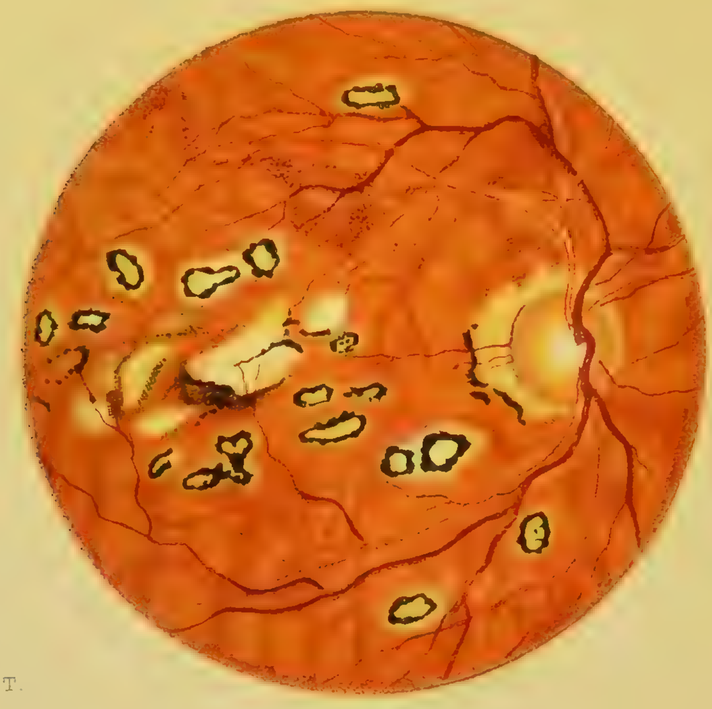

FIG. 79.—Disseminated choroiditis.

While the characters just described enable one to distinguish the evident exudative from the evident degenerative stages, there are many cases where the diagnosis of the stage is by no means easy, and may be impossible. This is the case mainly where the principal changes seem to be limited to the pigment layer of the retina. The choroidal structure is then well seen in the area of the patches, and the main difference caused by time, is in the amount and arrangement of the pigment masses bordering them.

Various different processes, all of which are hardly, strictly speaking, inflammatory, may give rise to similar choroidal patches. There may be localised absorptions and proliferations in the pigment layer alone; proliferation leading to localised excrescences on the lamina vitrea, and associated with similar pigmentary changes; localised patches of atrophy in the chorio-capillaris, or of exudation in the stroma of the choroid. In a not uncommon variety of disseminated choroiditis, which variety seems usually to be a manifestation of hereditary syphilis, the patches are small, circular, and bordered by dense crescentic masses of pigment. In some cases of choroiditis the pigmentary changes are not limited to the region of the patches in the choroid, but are met with in the shape of irregular spots in the more superficial parts of the retina. Choroidal patches may form first in the region of the equator of the eye, and gradually invade more and more of the choroid, spreading towards its central portion, as well as anteriorly;

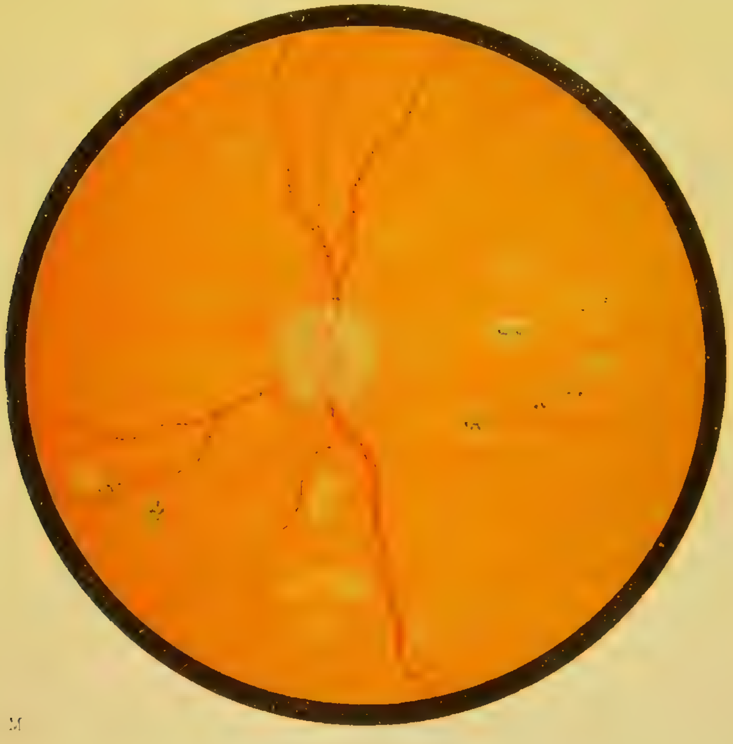

Fig. 80.—Disseminated choroiditis (recent).

or they may be confined to the region of the papilla and macula, a form of disseminatied choroiditis which is not uncommon, and to which Förster has given the name of *areolar choroiditis.*

It is hardly possible to say from the ophthalmoscopic appearances in any case of disseminated choroiditis to what extent the vision has been reduced. In many cases the most extensive ophthalmoscopic changes are met with where the visual acuity is found on examination to be hardly, if at all, subnormal. Often, however, in such cases, where the vision is found to be so good, there is some degree of night-blindness, and almost always the patient complains of a cloudiness in front of his eyes. The subjective symptoms are altogether more marked in the exudative than in the atrophic forms of the inflammation, the occurrence of fresh patches of exudation being accompanied by subjective sensations of light and colour, as well as by positive scotomata. The degree of visual disturbance and other subjective symptoms seems to depend in great measure on the site occupied by the patches of inflammation. Patches at or near the macula necessarily cause much greater interference with vision than those situated more peripherally. One frequently sees, for instance, in the case of the areolar form, the vision, which has hitherto been more or less good, all at once become very much deteriorated, without any marked change being visible in the ophthalmoscopic appearances. The reason of this is, that a fresh patch has formed at the fovea, or it may be, a previously existing change in that situation has so far altered as to involve the percipient elements of the retina, which up to that time have escaped. Central patches give rise at first to the appearance of distortion of objects looked at, or *metamorphopsia.* At a later stage this may be followed by a positive scotoma, or a blind spot, of which the patient is conscious, and which he projects in front of his eye as a clouded or black figure, the size of which increases with the distance of the plane of projection.

The scotoma is at first only seen in subdued light, and clears away as the surrounding illumination becomes greater, or the object fixed is brighter. In testing for positive scotomata, therefore, the test should be made in subdued light.

The metamorphopsia is due to a change, produced by the exudation or other alteration of the choroid, in the relative positions of the percipient elements of the retina, which causes a difference in the external projection of the impressions which they receive. In the case of a fresh exudation, the retinal elements are abnormally separated, so that a smaller number of elements come to occupy the same superficial area as before ; or, in other words, the same number of elements are spread over a larger area than under normal circumstances. The image of any object falling on such an area produces stimulation of a smaller number of retinal elements than it would otherwise do ; but the projection being unaltered, this is not compensated for, and the object appears diminished—that is to say, such changes give rise to *micropsia*. On the other hand, the shrinking resulting from the contraction of an old exudation, or from some atrophic change, may lead to a greater approximation of the retinal elements in the area involved, so that the same number of elements come to be spread over a smaller space than they originally occupied. Such an approximation gives rise, for similar reasons, to an unnaturally large apparent image, or to *macropsia*.

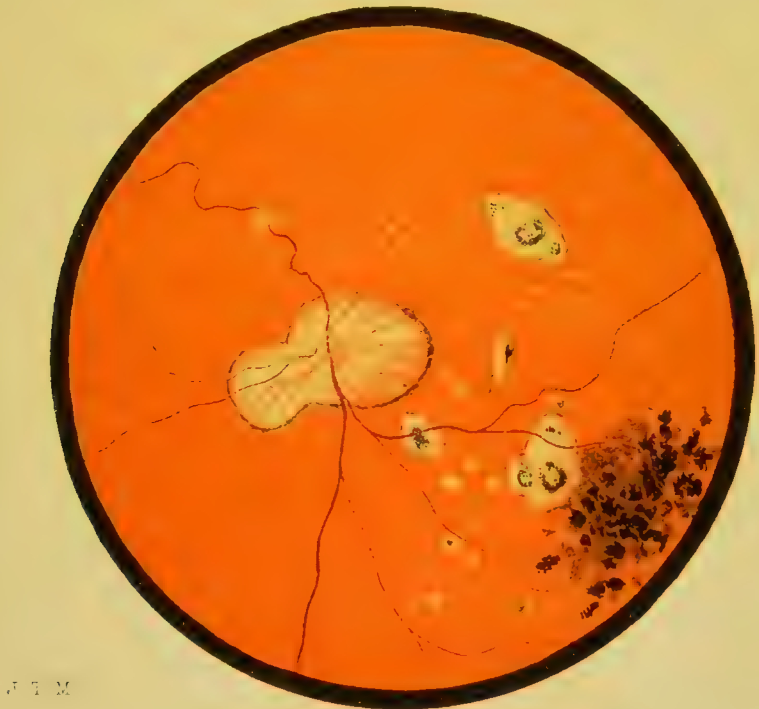

FIG. 81.—Disseminated choroiditis with marked pigmentary changes.

Förster, who was the first to explain the cause of retinal metamorphopsia, pointed out that the micropsia increases as the distance of the object from the eye becomes greater. In this way retinal is distinguished from accommodative micropsia. It is as well to bear this in mind, as a defective accommodation is not infrequently met with in cases where the choroiditis is acute, more especially in the syphilitic form.

The best method of testing whether or not any metamorphopsia exists (and it may be present without being spontaneously complained of) is to cause the patient to fix a mark in the middle of a number of parallel lines, a few millimetres apart, and to say whether they appear to him to run parallel, or to bend outwards or inwards at any particular place. In this way we may detect not only the existence of a localised metamorphopsia, but also whether we have most probably to do with a

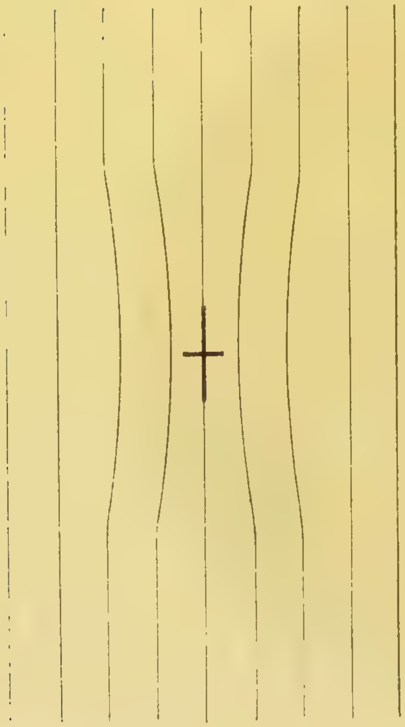

Fig. 82.—Showing distortion of parallel lines round point of fixation —micropsia.

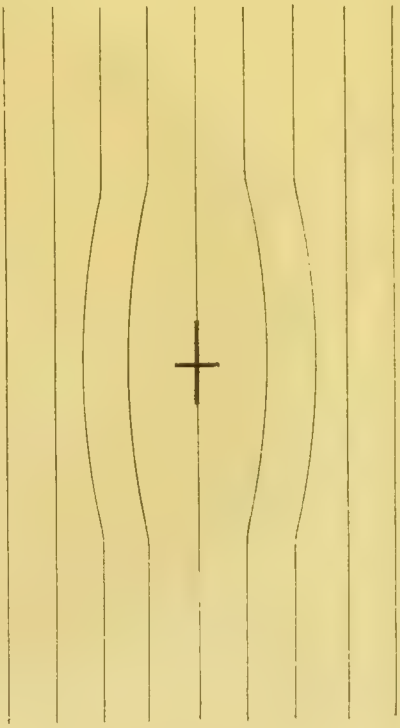

Fig. 83.—Showing distortion of parallel lines in the case of macropsia.

recent exudation causing a distension, or an old exudation or atrophic change which has led to a contraction and approximation of the retinal elements in the corresponding area.

Besides the defect in visual acuity caused by changes in the external layers of the retina, there may be other changes which have more or less influence on the vision, according to the site of the choroidal exudations. When the area attacked is in close proximity to the papilla, there is often found to be hyperæmia of the disc, with some interference with the transparency of the vitreous. This hyperæmia is the result of a participation by the scleral vascular ring in the congestion of the choroidal vessels. Again, an extension forwards of the inflammatory changes is apt to bring about a complication with cyclitis or even iritis. What the exact connection is between choroiditis and opacities of the vitreous is not very clear, more especially why such opacities should occur in some cases and not in others.

The *etiology* of disseminated choroiditis is very often obscure. A number of cases, and more particularly of the exudative variety, are probably syphilitic. It is very uncommon in children, in whom it is mostly a manifestation of inherited syphilis.

Treatment.—In the cases which are of syphilitic origin, anti-syphilitic treatment on the usual lines will often be found to be of use. But great harm may be done by treating other cases in the same way. This is more particularly so where the choroidal inflammation occurs in young people or comes on after some debilitating illness. Fresh air and moderate exercise with tonics, especially iron, are most useful in such cases. Cheerful surroundings and change of air are also to be recommended. It is certainly a mistake to confine such patients to a more or less darkened room, as is often done. They may be given dark glasses to wear outside, but should not as a rule be put under any more severe restraint. Wet packing for half an hour every morning sometimes appears to do good.

SENILE CENTRAL CHOROIDITIS.—When the vision of old people slowly but steadily deteriorates, and when on examination it is found that the field of vision is normal in extent in all directions, while with the ophthalmoscope the lens and vitreous are seen to be clear, a careful exploration of the macular region should be made. For this purpose it is generally necessary to dilate the pupil. The cause of this progressive defect will, in such cases, very often be found to be an alteration in the choroid immediately behind the macula.

If seen at an early stage the appearance presented by this disease is that of a reddish or reddish-yellow, usually irregularly oval-shaped patch, often only very slightly differing in colour from the rest of the fundus, but always presenting a very definite outline. Afterwards the patch assumes a more atrophic aspect, and the colour then presents a greater contrast to that of the surrounding parts; the edges become more irregular and bordered by pigment. The patch almost always appears in both eyes, though often when first seen it is further advanced in one eye than in the other. As a rule the patch is little more than half the size of the papilla, but it may be considerably larger.

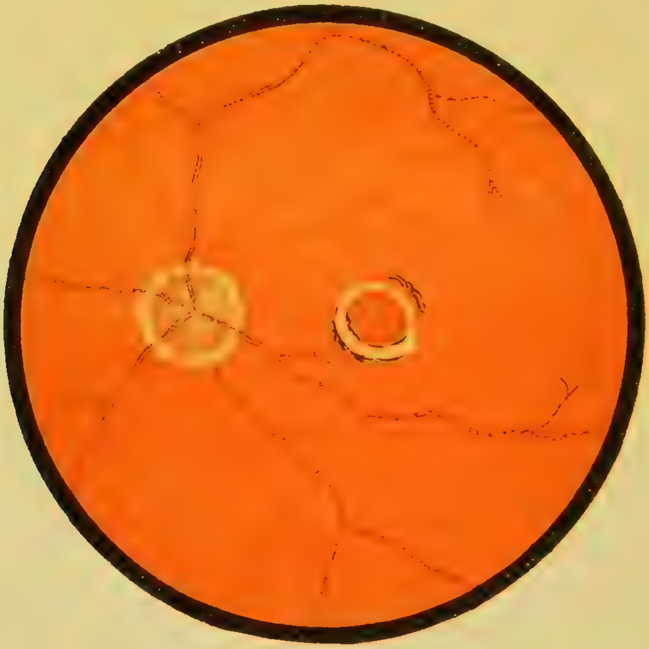

FIG. 84.—Senile central choroiditis.

The condition gives rise to metamorphopsia and to the appearance of a positive scotoma, of the existence of which the patient is more or less conscious. Central fixation is eventually abolished, so that the visual acuity is reduced to $\frac{20}{200}$ or less. Patients with central choroiditis are therefore unable to read ordinary print, but do not become quite blind, as there is no tendency for the condition to spread to other parts of the eye. On this account the *prognosis* is so far good, and it is therefore of considerable practical importance to make a correct diagnosis, and not confound the condition with any affection which may possibly proceed to blindness.

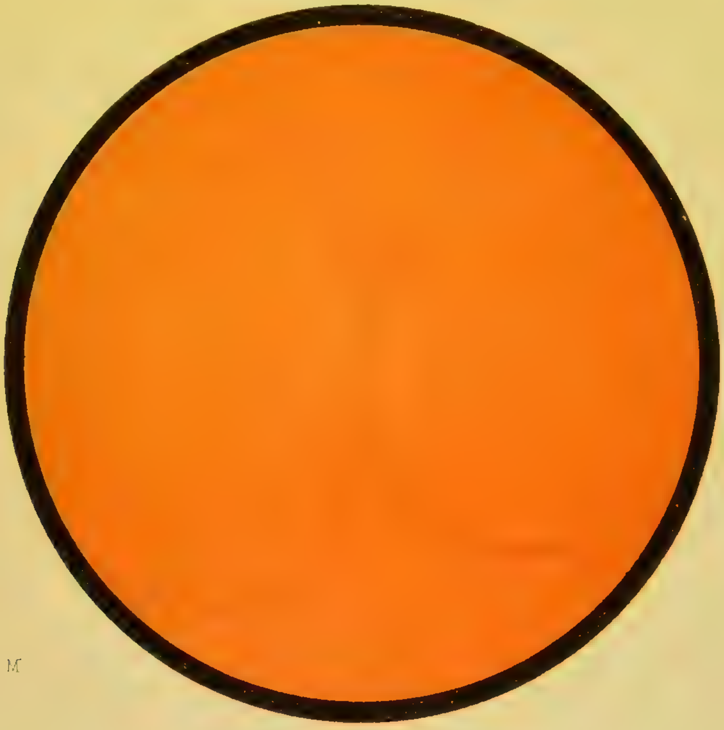

FIG. 85.—Acute diffuse choroiditis, with hyperæmia of disc and vitreous haze.

No *treatment* appears to have any influence whatever on this disease.

SYPHILITIC CHOROIDITIS.—While many changes recognised with the ophthalmoscope as choroidal are more or less directly the result of syphilis, there is one form of acute inflammation which, if not invariably a manifestation of syphilis, is so at all events in the vast majority of cases. This form seems first to have been described by Jacobson as syphilitic retinitis, and some authors still retain the name. Förster however, pretty definitely demonstrated the true choroidal nature of the affection. It is a late secondary or early tertiary symptom of syphilis. In more than half the cases met with, traces of other secondary manifestations are found at the time of the outbreak of the choroidal inflammation. It is most frequent in elderly individuals, and appears altogether more likely to make its appearance in cases where syphilis is acquired late in life. Sometimes the choroiditis follows after a few months on an attack of iritis. It is mostly met with in both eyes.

The *objective symptoms* are often very slight, viz., hyperæmia of the disc, with faint haze of the surrounding retina, and at the same time a fine diffuse opacity in the vitreous. The hazy appearance of the retina is partly due to the veiling by the opacity in the vitreous, which is always most dense in the central portion, and partly to serous exudation in the retina itself. It clears up to some extent as the eye remains in one position during the examination, owing to the sinking down of the vitreous opacities. In a number of cases the opacities are much more dense, and render the details of the fundus more or less indistinct. In all there is a great tendency for them to continue for a long time, or to disappear and reappear at intervals. In short, the constant association with more or less dense opacities of the vitreous is one of the characteristic symptoms of this disease.

The diminution in vision varies much in different cases, and the acuity does not altogether appear to correspond to the ophthalmoscopic changes. When the vision is very defective—less than $\frac{20}{200}$—the vitreous opacities are probably always dense. The defect is often found to be relatively greater in the more central portion of the field of vision than it is towards the periphery. Often a ring or belt-shaped area of the field, which is functionally normal or nearly normal, may be found surrounding the defective central area. The ring may either be complete or interrupted in one or more places. Considerable differences, however, occur in the shape of the field of vision.

Very characteristic of this disease is an enormous difference in the acuity of vision, as well as in the form of the field of vision, found on examination at one time in a feeble, at another in a strong illumination. There is in fact often most marked night-blindness. Thus one frequently finds that a patient who cannot read even large print when still sufficiently illuminated not to cause any perceptible diminution of acuity in the vision of the normal individual, is nevertheless able, when the illumination is strong, to read, though it may be not without some difficulty, the very smallest print. He requires a more powerful light than is necessary under normal conditions to evoke the full functional activity of the retina. On the other hand, when the light is strong enough, differences in degree of intensity may be appreciated in a manner which does not greatly differ from the normal.

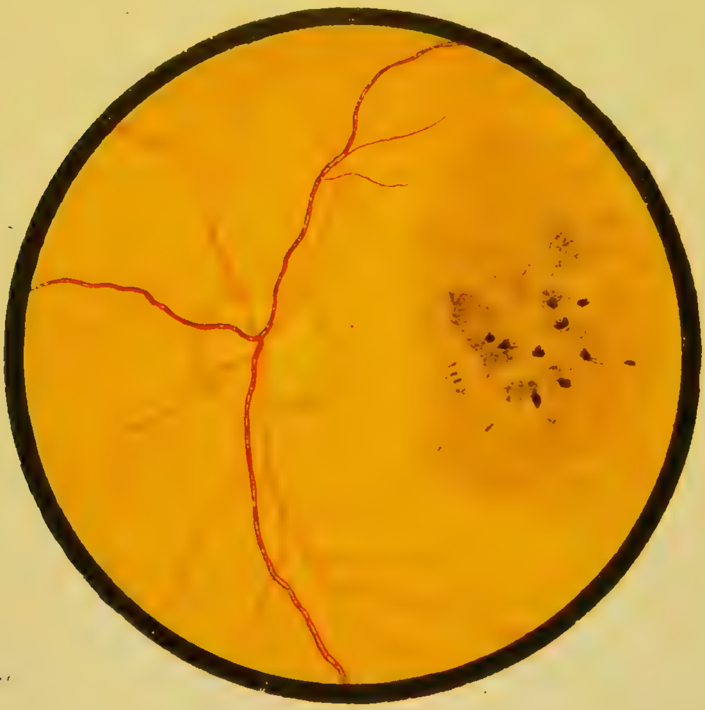

Fig. 86.—Choroiditis, with marked disturbance of retinal pigment.

This form of defect of the light-sense is more or less characteristic of all choroidal changes, and is one reason for considering the disease in question to be essentially an inflammation of the choroid, and not of the retina. The presence, too, of opacities in the vitreous, the occasional association with iritis, as well as the subsequent ophthalmoscopic changes met with in the more severe cases,—all characterise the inflammation as choroidal.

Subjective sensations of light are often complained of, and are sometimes painfully persistent. They originate in those parts of the retina which are affected by the subjacent inflammatory changes, and are intensified by strong light, or by anything which temporarily increases the action of the heart. Such subjective light-sensations do not render the vision any worse; although they may be projected over an object looked at, they do not interfere with its distinctness.

Metamorphopsia, and especially micropsia, is a very frequent symptom of acute syphilitic choroiditis. The micropsia is often extremely marked, and more so for objects held at some distance from the eyes. It is most apparent to the patient where the choroiditis exists in one eye only.

Acute syphilitic choroiditis may be recovered from without leaving any trace, and with perfect restoration of vision. More frequently some defect of vision remains, due to the persistence of opacities in the vitreous and the gradual development of changes in the choroid, which have much the appearance of other forms of disseminated choroiditis. Occasionally very dense opacities remain, and these cases are usually associated with inflammatory changes in the iris and ciliary body as well.

When considerable defect in vision remains after the first month or two from the beginning of the inflammation, ophthalmoscopic changes are sure to be met with in the choroid. One form which these changes may assume has very much the appearance of retinitis pigmentosa.

Typical cases of retinitis pigmentosa are, however, quite different from the variety following severe syphilitic choroiditis, and are easily recognised by the delicate shapes of the pigment deposited in the retina, its relation to the vessels, and the absence often of any disappearance of the pigment of the hexagonal cells, and always of any marked atrophic changes in the vessels of the choroid. Cases of true retinitis pigmentosa then, in which the pigment is scattered over a fundus which reflects a uniform red or reddish-yellow light, and in which the structure of the choroid is more or less completely hidden, cannot possibly be mistaken for the degenerative form which follows choroiditis. It cannot be denied, however, that in some less typical cases the differential diagnosis between the true and what may be called the syphilitic or spurious variety is not without difficulty. Probably in the syphilitic type the deposits of pigment are never as delicate in form, and the choroidal changes, more especially in the way of atrophy of the vessels, more complete, while at the same time the relative perfection of the central as compared with the peripheral vision, where the blindness is not complete, is less pronounced.

The best *treatment* is, no doubt, the mercurial, and preferably by inunction, continued as long as possible without giving rise to stomatitis, a method of treatment already fully referred to in connection with syphilitic iritis. At the same time, it would appear to be of not a little importance to keep the patient in semi-darkness. This may be done by making him use tolerably dark smoked glasses, of such a shape as to exclude light coming from the sides, as well as from the front. He should at the same time avoid stimulants and any violent exercise, and be altogether prohibited from making any attempt at reading, or using his eyes for any work necessitating accommodation, or a strong illumination.

ANTERIOR SCLERO-CHOROIDITIS.—In this variety of choroidal inflammation, there is a participation of the overlying sclera in the inflammatory process. Frequently this leads to a weakening of the portions of sclera involved, and a consequent staphyloma, or bulging, due to the yielding which then takes place to the pressure from within the eye. The changes which occur in the choroid cannot be seen with the ophthalmoscope, as they are confined to that part which lies in front of the equator of the eye.

The disease occurs in an acute and a chronic form, and may accordingly give rise to very different changes. Even acute cases may go on for many months before the eye comes to rest, while the chronic form lasts for years.

Acute anterior sclero-choroiditis generally attacks a circumscribed portion of the sclera and choroid, in the neighbourhood of the iris, but the complete zone surrounding the cornea may be involved. The subconjunctival tissues over the site of the inflammation become hyperæmic and swollen, just as in the more superficial episcleritis, though there is less tendency to localisation. The pain, too, is pretty severe, while episcleritis is remarkably painless as a rule. The infiltration soon spreads to the cornea, and often to the iris as well, giving rise to synechiæ, and other changes characteristic of iritis. The infiltration in the cornea may be so dense that, when the inflammation has subsided, it often looks just as if the sclera had encroached on the cornea, a condition sometimes spoken of as *sclerotising keratitis*, which abolishes the natural regular border between the cornea and sclera. In old-standing cases, in which there have been repeated attacks of the inflammation, the margin of the clear cornea becomes in this way extremely irregular. In very severe, diffuse, long-continued cases, the sclerotising of the cornea may be complete, and the whole anterior part of the eye thus become opaque, and more or less flattened.

In the *chronic form* of the inflammation, the inflamed area undergoes atrophic changes, and becomes thinned. It then gradually assumes more and more of a bluish or greyish-blue colour ; and as at the same time there is often an increased secretion and abnormally high tension in the eye, as the result of the associated iritis, this discoloured area slowly begins to yield and bulge forwards. This gives rise to variously shaped protrusions, or staphylomata. The staphylomata may form at the corneo-scleral margin, over the ciliary region, or somewhat further back, or all these parts may be at the same time involved in the protrusion.

There is rarely much, if any, pain experienced by this gradual distension, which may occupy years in arriving at any great size, and generally takes place after the vision has been very considerably reduced. In the acute cases there is pain, both spontaneous and on pressure, over the inflamed area. From the stretching of the retina, too, patients complain of subjective sensations of light.

One marked peculiarity of the disease is its intermittent character. Different portions of the anterior part of the choroid and sclera are affected on successive attacks. It appears to be considerably more common in women than in men, and is most liable to occur before the age of full growth is attained. It is believed by some to be connected with uterine disorders. Undoubtedly, in some cases, such a connection exists, but in my experience this is more frequently not the case. Chronic arthritis is not uncommonly met with in patients suffering from this disease.

The *prognosis* is always bad, and worse in the chronic than in the acute form.

In the *treatment*, any state of the general health which appears likely to have a bearing on the local affection must be attended to. The digestive functions more especially require attention. Anti-rheumatic treatment will often be useful, where there are distinct rheumatic manifestations elsewhere. In such cases a visit to Harrogate, Buxton, Wildbad, or Marienbad, may do good. Antiphlogistic treatment is required for the local condition when it presents itself in the acute form. Leeching, mercurial inunctions, pilocarpine injections, &c., are then of more or less service. At the same time, as complete rest as possible must be given to the eyes, and dark glasses constantly worn.

Besides the medical treatment, the question of surgical interference has often to be considered. Operations must not, however, be lightly overtaken, as they may produce immediate or subsequent bad results. Thus, if an iridectomy be performed in some old-standing cases where there has been thinning, vitreous may escape in considerable quantity, owing to fluidity, or to disease of the zonule, and be followed by serious consequences. The operation is indicated in cases where the tension is markedly increased, and in which there is not any excessive staphylomatous change. In all cases, great care must be taken to obtain a speedy healing of the wound, and to avoid any catching of the iris in it. In some of the more persistent cases of the acute sclerotising form, I have obtained very satisfactory results by performing peritomy; in other cases, apparently very similar, this operation is of little use. When the eye has come to rest after peritomy, an iridectomy may be done as well. This seems advisable as a prophylactic measure, considering the great tendency there is to recurrence. It is all the more called for when synechiæ have resulted from the accompanying iritis, but the contra-indications, already referred to, must be borne in mind.

POSTERIOR SCLERO-CHOROIDITIS.—*Staphyloma Posticum.*—A similarly slow, degenerative, inflammatory change of the choroid, with thinning of the corresponding portion of sclera, takes place at the back of the eye. Staphyloma posticum is mostly found to the outer side of the papilla, but may extend all round it and involve the macula as well. Just as in the anterior form, this inflamed area yields to the intraocular pressure. In this way a true staphyloma posticum is produced. The name staphyloma posticum is given, however, to very varying degrees of defect in the choroid in the immediate neighbourhood of the disc. The defect may be merely due to the aperture in the choroid at the site of the entrance of the optic nerve being larger than that in the sclera, whereby the scleral ring becomes broadened to the outer side, showing as a crescentic white patch with its concave margin next the disc. Such a crescent may be met with along with all states of refraction, but is more frequently seen in myopic eyes. A very great enlargement of this patch may take place as the result of a slow form of sclero-choroiditis. This low inflammatory change only takes place in myopic eyes. A similar appearance due to atrophic thinning of the choroid is met with as a senile change.

As the area of degeneration extends in a myopic eye, it comes more and more to encircle the disc, though it is usually, even when very extensive, most marked to the outer side. In a small proportion of cases, the crescent lies at the lower margin of the disc. The white colour of the staphyloma posticum is due to reflection from the sclera below the patch of atrophy of the choroid and pigment layer of the retina. The superficial layers of the retina are not as a rule implicated in the process, and as a consequence

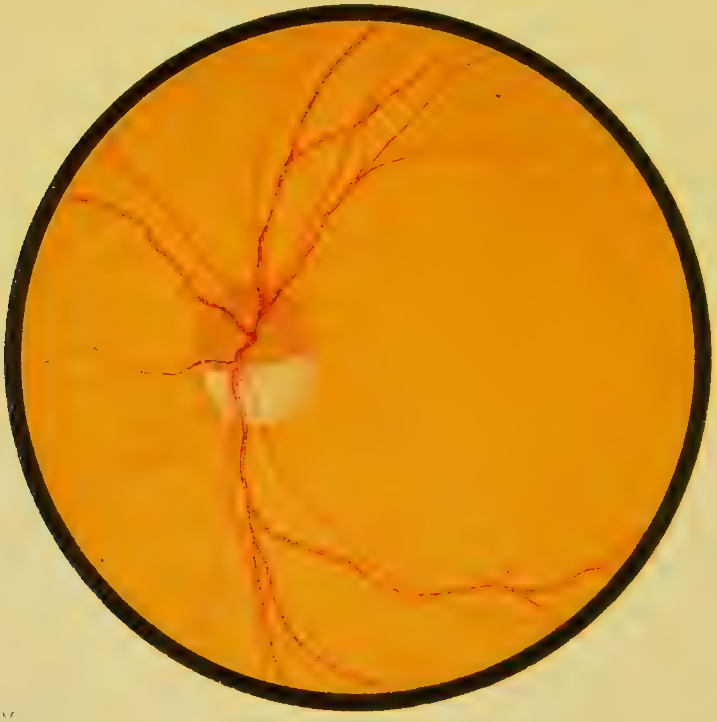

FIG. 87.—Case of staphyloma posticum at lower margin of disc.

of this, the retinal vessels may be seen to course over it, just as in the cases of choroiditis elsewhere.

The margin of the staphyloma which is farthest away from the disc may be sharply defined, and is then, usually at the same time, the site of a greater or less pigmentation. In other cases the patch merges at this situation into the surrounding fundus without showing any very decided definition. Speaking generally, the first condition is, an indication of an arrested, or only very slowly progressive change, while the latter should give rise to more suspicion, as showing a greater likelihood of progression. In the very high degrees of staphyloma posticum, the crescentic shape is altogether lost, and only a large white patch, sometimes with here and there some pigment spots, surround the disc on all sides. These large areas of degeneration may even involve the macula. The protrusion, by increasing the antero-posterior diameter of the eye, causes axial myopia. The greatest distension does not take place at the macula, and therefore the axial prolongation is not greatest in the line of vision. The myopia consequently, though progressive, does not increase at a rate which corresponds exactly to the increase of the elongation in the region of the diseased coats.

Posterior sclero-choroiditis, which causes the higher and progressive variety of myopia, has a tendency, just like the affection in the anterior part of the choroid, to remain quiescent for some time, and then break out afresh. It often causes a dull aching pain. This pain, and the evidence of progression in the degree of the myopia, as well as the defect in vision, which makes its appearance sooner or later, are the main signs of the disease being active. On the other hand, during a period of remission, the vision does not alter in acuity nor the myopia in degree, while at the same time the pain is absent.

This disease is common in the lower classes, so that the higher and most serious cases of myopia, unlike the more common form, in which the tendency to progression is not pronounced after full growth is attained, are found amongst the illiterate, as much if not more than amongst such as use their eyes much for reading, or for any work necessitating convergence of the visual axes. What the causes are seems to be altogether unknown. There is, besides, no evidence of any heredity, which is so important a factor in connection with the typical form of myopia. It appears as if various forms of malnutrition and constitutional weakness, inherited or acquired, predispose to it.

In connection with staphyloma posticum, and what must be looked upon as the immediate cause of the defect of vision in the progressive variety of myopia with which it is accompanied, are changes occurring in the region of the macula itself. These changes are rather different in their mode of origin from those characteristic of senile central choroiditis which they may afterwards come to resemble pretty closely. They frequently begin as a kind of irregularity in the pigmentation at the macula, which takes the form of differently shaped figures darker than the surrounding fundus. Gradually the centres of these figures exhibit a yellowish or atrophic coloration, which encroaches more and more on the darkened area, and eventually becomes an atrophic plâque very similar to those met with in disseminated choroiditis. It is not quite certain whether these macular changes are the result of atrophy from stretching alone, or

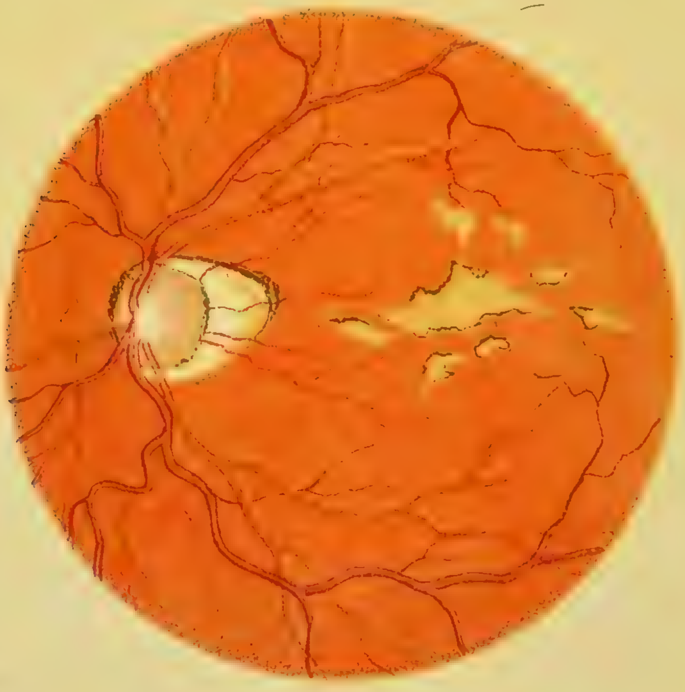

FIG. 88.—Staphyloma posticum, with changes at the macula. From a case of progressive myopia.

are caused by inflammatory exudations in the choroid as well.

Metamorphopsia is a constant symptom of this condition, and frequent complaints are made of muscæ, cloudy vision, and subjective light-sensations. The symptoms, in short, all point to irritation of the retinal percipient elements.

The *treatment* is, on the whole, very unsatisfactory, especially where the condition has been going on for some time. Reading should certainly be avoided, and dark glasses be more or less constantly worn at times when the subjective symptoms point to activity in the inflammatory process and progression of the myopia. At the same time it may be of use to leech or blister the temple. Horner has recommended frequent paracentesis of the anterior chamber, but of this treatment I have no personal experience. Iridectomy is certainly not of any use, and indeed not altogether safe. Other operations, such as tenotomy of the external recti, or even of all the recti, are probably never now performed, although at one time such treatment seems to have been pretty extensively practised, as it was held that the pressure of the muscles tended to increase the elongation of the eye.

A great deal too much has undoubtedly been made, and is still made, of the possible part which the muscles play in this respect. This has arisen in great measure from the erroneous idea that the elongated globe moves less freely than it really does round its axis of rotation, an idea which seemed to be supported by the mistake of taking insufficiency of convergence, so common in high degrees of myopia, for insufficiency of the internal recti. A still more unwarranted assumption ascribes a *rôle* of importance in the etiology of progressive myopia to the action of the inferior oblique muscle. This view, which has been lately propounded by Landolt, has led him to suggest tenotomy of that muscle, which he performs, or proposes to perform, by dividing its tendon of origin.

PURULENT CHOROIDITIS.—A purulent inflammation of the choroid which may either remain confined to that membrane, or lead to inflammation of all the tissues of the eye—*panophthalmitis*, occurs as the result of various lesions. Thus it may be set up by operations performed on the eye, or by penetrating wounds, with or without the lodging of foreign bodies within the eye. It may follow fresh perforations of the cornea, or any accident which causes a bursting or inflammation of a staphyloma of the cornea or sclera.

In a small but important group of cases the etiology is of an altogether different nature; in such the purulent choroiditis may result from thrombosis in the ophthalmic veins, from embolism in the choroidal arteries, or from septicæmia. The first class of cases may be grouped as cases of traumatic purulent choroiditis, the second as metastatic purulent choroiditis.

Traumatic Purulent Choroiditis.—The symptoms of purulent choroiditis are—very great pain in the eye, accompanied by rapid and complete loss of vision, redness and swelling of the lids, and chemosis. Swelling of the orbital tissues is also more or less marked, causing a tendency to protrusion of the eye, the mobility of which is at the same time interfered with, partly on account of the participation of the muscles and the mechanical difficulties which the presence of the exudation occasions, and partly, no doubt, owing to the pain alone. In cases which go on to panophthalmitis all these symptoms are more intense, and at the same time the cornea becomes more and more hazy from infiltration of leucocytes; the aqueous humour becomes muddy and purulent, and iritis with hypopyon makes its appearance. Purulent choroiditis always leads eventually to shrinking of the eye. This may follow rapidly on a spontaneous perforation of the coats and the discharge of pus externally, or it may more slowly result from consolidation and partial absorption of the purulent matter without perforation. In such cases, if the lens remain clear and the pupil be not occluded, the whitish mass of partially organised lymph may be visible.

In the *treatment* of purulent choroiditis we cannot expect to save the sight of the eye. All that can be done is to alleviate the excessive pain from which the patient suffers. For this purpose, when vision has been lost, poultices may be freely applied, and an opening made in the eye to admit of the discharge of the pus. The opening may be made right through the cornea, thus allowing the lens to escape, or it may be made, as some advocate, in the sclera. Instead of poulticing, the eye may be frequently syringed out with a solution of corrosive sublimate. This, which has been recommended by Chibret, seems sometimes to hasten the healing process. It is very useful when the suppuration has begun in the vitreous.

Enucleation should not be performed at the time of the inflammation, owing to the risk that there is of thereby setting up meningitis. Meningitis occurring after enucleation performed for panophthalmitis is certainly rare, and when it does occur, it seems probable that it is the result of the transference of septic matter along the lymph channels of the eye, or tissues of the orbit, to the membranes of the brain. When the inflamed eye is left alone, or merely incised to give exit to the pus it contains, this transference is much less liable to occur, if indeed it ever does occur. The reason of this is no doubt that a natural limitation takes place between the healthy and necrotic tissues, whereas the removal of the eye causes the opening up of lymphatic and vascular channels, and brings a comparatively healthy wounded surface in more immediate contact with the inflamed tissues. Thus it is found that the danger is greatest in the case where the pus from the eye which is being removed is allowed to come in contact with the wound made. This may happen when the globe is perforated by the scissors during the operation, or has spontaneously burst before the enucleation is begun. Again, when meningitis has occurred, it has always come on within forty-eight hours or so after the operation. Whether the risk of enucleation in panophthalmitis can be altogether overcome by antiseptic precautions, or not, is certainly doubtful; at all events most rigid antiseptic measures are called for if enucleation be practised. Possibly less danger attaches to the operation of evisceration. The wound produced by this operation is less severe, and can be more effectually washed out by antiseptic lotions. For these reasons evisceration is probably preferable to enucleation, but sufficient experience of the operation has hardly yet been gained to admit of a definite conclusion on this point. Truc has recommended evisceration followed by enucleation. The object of this is to remove the septic matter contained in the eye before opening up the cellular tissue of the capsule. If enucleation be performed at all, this would seem, theoretically at all events, to be a good precaution.

The question as to whether enucleation should be done or not at a later period when the eye has shrunk, and the symptoms of active inflammation have subsided, is one which, as a general rule, may be left pretty much to the patient, as there is little danger of sympathetic inflammation occurring in the other eye.

When purulent choroiditis seems to be threatened, it would appear that it sometimes can be kept off by the application of a tight bandage over the eye.

Metastatic Purulent Choroiditis.—By far the greatest number of cases, in which a meningitis from which the patient recovers is accompanied by purulent choroiditis, occur in very young children. Often such cases are first seen by the ophthalmic surgeon after the meningitis has subsided. The inflammation in the choroid leads to shrinking of the globe, never to panophthalmitis. And the appearance which the eye then presents is that which in this country is often called *pseudo-glioma.* The iris is then muddy, more or less atrophied, and pressed forward by the lens, so that the irregular pupil comes to lie close up to the clear cornea. The periphery of the iris is, however, in most cases retracted, sometimes markedly so. Through the lens, which usually remains clear, a white mass can be seen lying in the vitreous. The tension of the eye is diminished, and there is complete blindness.

It is seldom that any difficulty can arise in the differential diagnosis between this condition and true glioma, which also occurs in children; the retraction of the iris at the angle of the anterior chamber, and the diminished tension, are sufficiently characteristic symptoms when present.

Choroiditis following meningitis is probably due to direct transference of inflammatory products along the sheath of the optic nerve. One or both eyes may be affected, more frequently only one. There appears to be no danger of sympathetic inflammation of the other eye where one eye alone is affected, and therefore enucleation is not necessary. The evidences of the meningitis are not always equally marked. Sometimes there have been distinct convulsions, at other times only loss of appetite and drowsiness, with the history of sudden redness and swelling of the eye, and a yellow kind of glimmer from the interior. This appearance, with the chemosis and hypopyon, and the evident interference with vision, distinguish the cases from simple iritis.

Purulent choroiditis in one, sometimes in both eyes, leading to complete destruction of sight and to shrinking of the eye, either from perforation, or absorption and organisation of the deposit in the vitreous, may occur in pyæmia, puerperal fever, erysipelas, and suppurative endocarditis, as well as in connection with inflammations, both idiopathic and traumatic, about the head or face. The immediate cause of the inflammation in these cases is probably not always the same, yet it is often, no doubt, as has actually been demonstrated, due to embolic infarcts in the choroidal vessels, and these have been shown by Hosch to contain micro-organisms. In other cases, again, such as those which occur after debilitating fevers, some other cause probably exists,—sometimes, it may be, thrombosis of the choroidal veins.

In cases, again, of thrombosis of the cerebral sinuses, we may find a pretty sudden protrusion of the eye with amblyopia or amaurosis, a dilated and motionless pupil, and more or less chemosis of the conjunctiva and œdema and redness of the lids. This may occur on both sides, but is most frequently found on one side only. Probably the thrombosis in the sinuses is not sufficient of itself to give rise to the choroiditis, but must be associated with thrombosis in the ophthalmic veins as well. Such cases always end fatally. The process may begin in the sinuses of the brain and spread to the ophthalmic vein, or it may result from a phlebitic process in the neighbourhood of some inflammation in the face, and spread back to the sinuses. In the first case there may often be great difficulty in diagnosing between the thrombosis of the sinuses and meningitis, as the cause of the panophthalmitis. When it occurs on both sides, more especially with an interval between the appearance of the inflammation in the two eyes, thrombosis would appear to be most likely. The state of the pupil may sometimes be of diagnostic importance in this respect,—in thrombosis it is generally dilated and motionless while retaining its normal form; in choroiditis from meningitis, on the other hand, it may be irregular and bound down by synechiæ.

SEROUS CHOROIDITIS.—A purely serous inflammation of the choroid alone is generally supposed to exist, and to produce all the symptoms of glaucoma. It does not present any characteristics which call for special reference here. It is often associated, however, with serous irido-cyclitis, and may then come on after severe illness, more particularly after recurrent fever.

A peculiar form of choroiditis is sometimes met with, in which a very copious exudation takes place in the region of the macula or the papilla, leading to the formation of a flat prominence in this situation. In the cases I have seen, I have always been in doubt as to whether the affection should not be looked upon more as a detachment of the choroid, rather than an inflammation pure and simple of the membrane. The disease, whatever be its exact nature, is an extremely serious one as far as the sight of the eye is concerned.

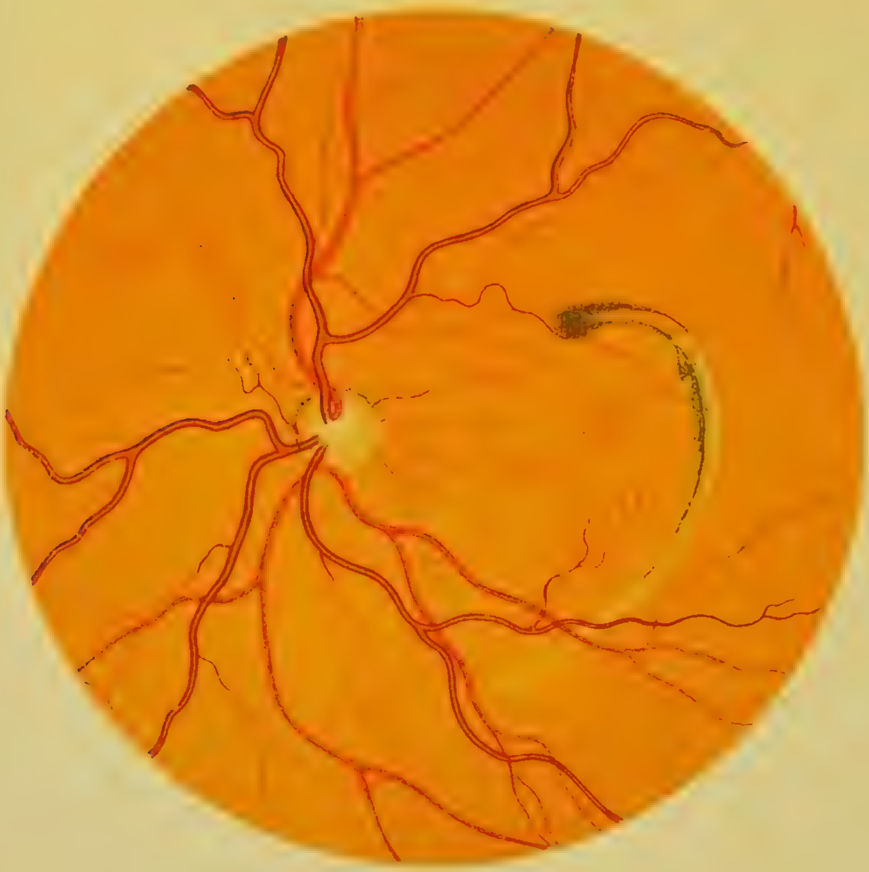

FIG. 89.—Rupture of the choroid (inverted image).

RUPTURE OF THE CHOROID.—The choroid may be ruptured, partially or completely, by a blow on the eye with some blunt object, and with or without there being a rupture of the retina at the same time. The same injury may give rise to retroversion or separation of the iris, dislocation of the lens or other lesions in the anterior segment of the eye, but often the rupture of the choroid is the only change produced. The sclera immediately behind the ruptured choroid never gives way.

Ruptures of the choroid take place near the posterior part of the eye, and possibly at the anterior part, where they cannot be seen with the ophthalmoscope. They may be to either side of the optic nerve, or above or below it, and almost always coincide very nearly with segments of circles described round the papilla as a centre. Bifurcations may occur, and in some instances two distinct and concentric ruptures may take place. One such case has come under my own observation, in which the rupture near the papilla was considerably smaller than the other, while the mid-point of both lay pretty nearly in the same meridian (see Fig. 90).

The ophthalmoscopic appearances vary according to the completeness of the rupture and the length of time which has elapsed since the accident. If the eye be examined immediately after the injury, the rupture cannot, as a rule, be seen, but the retina is found to be swollen and hazy, and more or less veiled. In a few days this haziness in the retina disappears, and a sharply cut linear yellowish figure, concentric with the papilla, and over which the retinal vessels are seen to pass, makes its appearance not far from the papilla. This gradually becomes more and more white, and in the course of time is here and there pigmented. Pigmentary changes also often develop in other parts of the surrounding retina, particularly in the immediate vicinity of the optic disc. When the rupture has been incomplete, the colour which it presents on ophthalmoscopic examination is never so white, and is sometimes only slightly different from the surrounding fundus, so that the diagnosis may be a little difficult. Rupture of the choroid generally causes very little bleeding, but the nature of the lesion may remain for some time undetected, owing to the bleeding from the iris, which has been injured at the same time.

The extent of the injury to vision differs very much in different cases, depending on the position and extent of the rupture. In large ruptures the vision is always permanently much reduced. Even small ruptures, if they take place immediately behind or close to the macula lutea, cause great interference with sight. In almost all cases the vision is much reduced immediately after the accident, and gradually improves to a greater or less extent as the œdema of the retina passes off. Sometimes it would appear that the vision, after recovering to some extent, again slowly deteriorates, as the result probably of changes taking place in the optic nerve.

When the retina gives way as well as the choroid—an accident which is of very much rarer occurrence—the evidence of surrounding hæmorrhages is more marked, and one misses the retinal vessels crossing any part of the white sclerotic background laid bare by the separation of the membranes. Retinal vessels are, on the other hand, seen here and there to curl round the margin of the white space, and then continue their course in the uninjured retina.

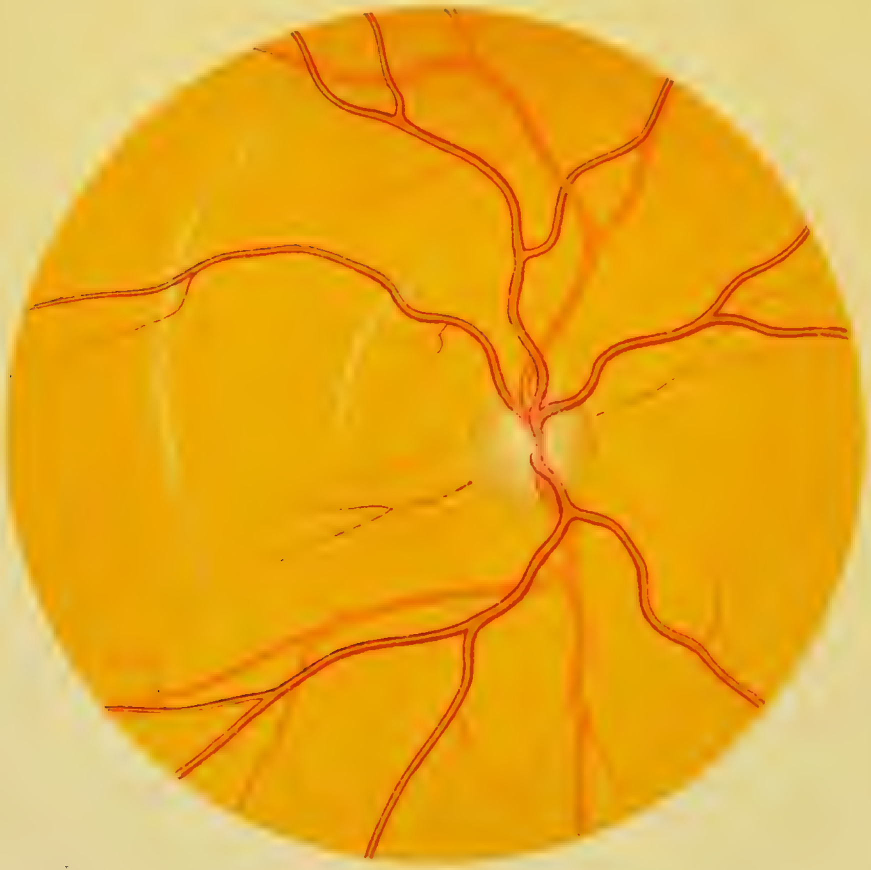

Fig. 90.—Double rupture of the choroid (erect image).

The mechanism of rupture of the choroid has not as yet been quite satisfactorily explained. It used to be supposed that, owing to the firmer attachment of the choroid at the posterior pole, where so many vessels pass into the eye, the dragging, which momentary alteration in the shape of the eye produced by a severe blow must cause, would act more on this unyielding portion than anywhere else. This explanation is unsatisfactory for several reasons. In the first place it does not account for the invariable direction of the rupture with its concavity towards the optic papilla. Again, the numerous experiments which Voelckers, Berlin of Stuttgart, and others have made on animals, with the object of producing rupture of the choroid, have either altogether failed or have given rise to a lesion that cannot be said to be exactly analogous to that met with clinically. It could not possibly hold good either in the case of a double rupture, when the two lie across the same meridian of the fundus, such as the one I have figured. It is more probable, therefore, that another explanation, given by Becker, comes nearer the truth. Becker supposes that at the time of the blow the nature of the distortion which the eye suffers is a pressing in of the optic nerve towards the middle of the eye, producing a fold or a series of folds in the coats immediately surrounding the papilla. At these folds both retina and choroid are stretched, but the former, being more elastic and less firmly fixed, yields, whereas the latter, from the nature of its attachments, gives way. He bases this view partly on the ease with which this distortion can be produced in an eye removed from the body, and partly on the failure of the experiments on animals, which may then be accounted for by the fact that in so many the relation of the optic nerve to the back of the globe is different from that which exists in man. The subsequent pigmentation occurring round the disc in some cases is also suggestive, as the immediate neighbourhood of the end of the nerve must no doubt suffer by the injury. Possibly in some cases the very opposite may take place, and the nerve, instead of being forced into the eye, may be more or less driven away from it.

COLOBOMA OF THE CHOROID.—Just as in the case of the iris an arrest of development may lead to the non-closure or imperfect closure of the fœtal fissure, so in the choroid a similar defect may result from the same cause.

Coloboma of the choroid is most frequently associated with coloboma of the iris, but may be present alone, and be found in one or both eyes. Other evidences of arrested development are sometimes met with in the cases in which this condition of the choroid is found, as, for instance, hare-lip and cleft palate. A more frequent concomitant condition is microphthalmos, in which the whole eye is imperfectly developed.

As in the case of many other defects of the same nature, the occurrence of coloboma of the iris and choroid is often ascribed by mothers to emotional disturbances experienced during pregnancy, most frequently to their having been impressed by the sight of some individual who has been blind, or exhibited some deformity in the region of the eye. How far such beliefs have any foundation in fact, it is impossible in the present state of our knowledge to say.

The true coloboma of the choroid is always met with downwards, or downwards and inwards, just as in the case of coloboma of the iris. The defect is usually narrower in the region of the papilla, which may or may not be included in it, and broader towards the periphery. When a coloboma exists in the iris as well, the two may be continuous, owing to a defect in the ciliary processes, or,—and this appears to be more frequent,—they may be separated by a bridge of normal, or apparently normal, ciliary structure; the anterior ending can then often be seen on ophthalmoscopic examination. Sometimes one or more normally

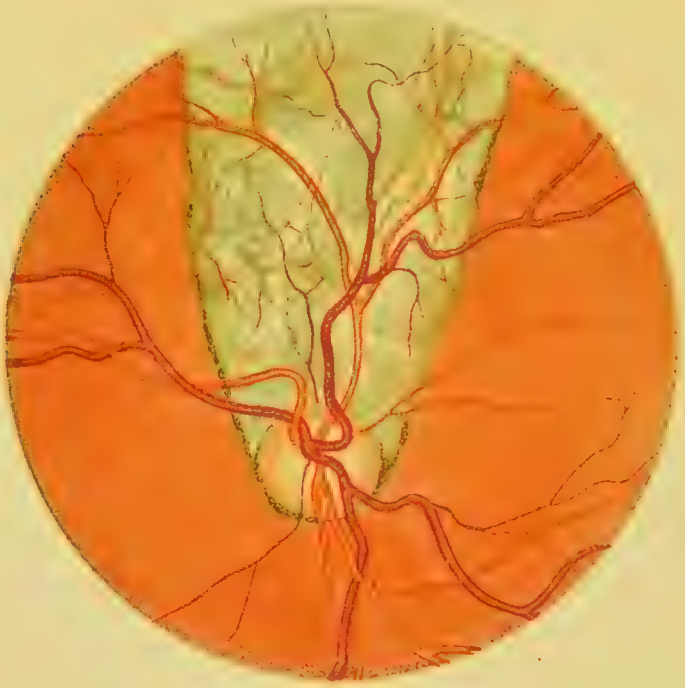

Fig. 91.—Coloboma of the choroid (inverted image).

pigmented portions of the choroid bridge across the coloboma which is thus separated into two or more portions. In such cases the defective islets may be broader than they are long. Occasionally, too, smaller guttate portions are seen to either side of the main defect.

In all cases the retina is either completely absent, or only very imperfectly developed in the region of the coloboma. There is therefore always to be found, except apparently in the very rare cases of coloboma in the region of the macula, which are only doubtfully of the same nature, a more or less complete scotoma, corresponding in extent to the coloboma. Over this area of the upper portion of the field there is either absolute blindness—that is to say, an inability to distinguish the strongest light from darkness—or a certain degree of perception of light. The latter seems to be the more common. More or less defect of vision altogether is also almost invariably found in eyes with coloboma of the choroid. Besides the retina and choroid, the sclera is also not altogether free in some cases from participating in the defect; it is thinned and occasionally staphylomatous.

On ophthalmoscopic examination a brilliantly white reflection is seen from the region in which the retina and choroid are defective. The surface of the coloboma is never of an equable colour throughout. Here and there, there are often to be seen masses of pigment. The white area, too, is often broken up by a number of different sized, very ill-defined, faintly bluish, spots, corresponding to irregularities in the sclera, which is laid bare by the absence of the choroid, and the reflection from which causes the marked, almost dazzling, whiteness which is presented by the ophthalmoscopic picture of a coloboma of the choroid.

Vessels are also seen to course across the white area in different directions. These are both ciliary and retinal vessels. In many cases of coloboma, only fine and narrow branches of the retinal vessels cross the coloboma, and in some there is an absence of retinal vessels altogether. This depends entirely, no doubt, on the degree to which the defect in development has involved the retinal structures which lie anterior to the pigment layer. There is no difficulty in distinguishing the retinal from the ciliary vessels. The former are distinctly seen to pass over the sharp margin of the coloboma as they come from the normal portions of the fundus; the latter, on the other hand, may be seen, after coursing over the defective area, and sometimes undergoing anastomosis, to pass on towards the choroid at the margin of the coloboma, where they disappear from view. When the disc is included in the coloboma, it may retain its normal appearance, but it is more often so altered as to be barely recognisable. It is usually very much enlarged, horizontally oval in shape, and its vessels appear to spring suddenly into view from its margin. No definite structure can be made out on its surface, there being a coloboma of the nerve sheath as well. A parallactic movement, indicating more or less staphyloma of the sclera in parts, or all over, may usually be detected.

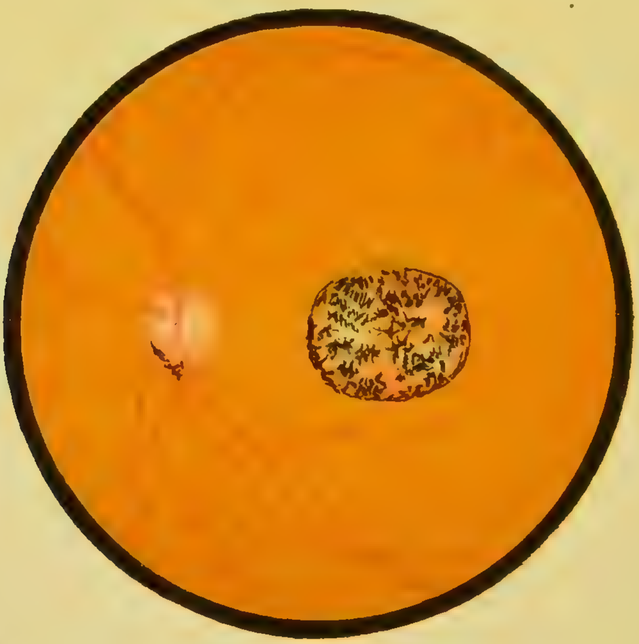

FIG. 92.—Coloboma at macula.

A rare form of coloboma, in which the defect occupies the region of the macula alone, to the extent it may be of only two to three diameters of the papilla, has been described by Streatfield and others. Some of these cases have probably not been true colobomata, but mere degenerative inflammatory changes which have taken place in intra-uterine or infantile life. Where the changes are bilateral and symmetrical, with clean-cut regular margins, as in a case which came under my own observation, there can be little doubt of their being of the same nature as other colobomata. In such cases there is apparently no scotoma to be found corresponding to the area occupied by the coloboma.

The nature of all forms of coloboma of the choroid is not as yet satisfactorily understood. The situation occupied by the defect, corresponding as it does to the fissure existing in fœtal life, renders it certain that the coloboma is in some way connected with that fissure. As the fissure only, however, involves the retina, including the hexagonal pigment cells which histo-genetically belong to it, we should expect to find the choroid intact, and only a coloboma of the retina as the result of its non-closure. Moreover, there can only have been a complete non-closure in cases in which all trace of the retina is absent in the coloboma, and in which no perception of light exists over the area in the field of vision corresponding to it. It has therefore to be explained, in the first place, why the choroid participates in the abnormality; and secondly, why such a definite arrest of development takes place in this situation, even when the retinal fissure must be supposed to have closed. In the cases which have been examined anatomically, the place of the choroid has usually been found to be occupied by connective tissue, but there is no evidence to show whether this has been of a cicatricial formation or not. All that can be said is—(1.) That when an arrest of development leads to non-closure of the retinal fissure, the underlying choroid, as well as the sclera in some instances, has its development more or less interfered with; and (2.) that there seems to be a tendency, apart from the non-closure of the fissure, to the occurrence of defective development of all the coats in its vicinity. Whether these changes are the result of intra-uterine inflammation, or merely of some circulatory abnormality, cannot be said with any degree of certainty. The first is rendered probable, not only on account of the appearances which have been met with anatomically, but also from experiments performed by Deutschmann on animals in which destructive processes produced in the eyes of the mother have led to defects of different kinds, amongst others colobomata, in the offspring. The etiology of the small central colobomata is still more uncertain. In the true cases the vision has been found not much if at all reduced. In one case which I tested by Bjerrum's method (see Chap. I.), I was unable to detect any visual defect. Possibly there may be some light-sense defect, but so far as I am aware the light-sense has never been tested in these cases. As it is certain that the rods and cones, or the principal percipient elements of the retina, are intact, it seems probable that the hexagonal cells are so also, but they are evidently devoid of their pigment. Cases occur in which a large circular staphyloma round the papilla is found in faultily developed eyes. These are apparently more of the nature of colobomata than patches of sclero-choroiditis, but their exact etiology is also unknown.

Only one case of total absence of the choroid, or *choroideremia*, has, so far as I am aware, been published. Mauthner, who observed the case, describes the appearance of the whole fundus as of a greenish-white colour, with here and there a patch of pigment. The retinal vessels could be seen coursing over the whole area. The defect occurred in both eyes, but more completely in the one; in it the vision was reduced to $\frac{5}{200}$, and a marked concentric restriction existed in the field.

Other cases of congenital restriction of the field, from extensive defects in the choroid, have been met with apart from the changes produced by intra-uterine choroiditis. Thus, Koenig has published a case in which this symptom, as well as night-blindness, was very marked, and the ophthalmoscopic examination revealed such a defective development of the chorio-capillaris that only at the macula and immediately surrounding region was there any reflex from the fundus. Such cases are all probably due to some form of arrest of development at an early age of fœtal life.

Tubercle of the Choroid.—Tubercle occurs in two forms in the choroid—as miliary tubercle and as larger tubercular masses. The first form is acute, and always secondary to tubercle in other organs. The second is chronic, and may or may not be primary. A distinction between the two varieties has been made by calling the former tuberculosis of the choroid and the latter tubercular choroiditis, but inasmuch as both give rise to inflammatory changes in the choroid, this distinction is hardly a very correct one.

Chronic Tubercular Choroiditis has no doubt long been observed, cases evidently of this nature having been described by the older writers on diseases of the eye, but it is only recently that such cases have been properly distinguished from other forms of tumour. The disease is certainly very rare, much more so than miliary tuberculosis. It occurs in one eye only, and appears sooner or later to be associated with similar disease of the brain, which ends fatally. The ophthalmoscopic changes in the cases which have been described have been those of optic neuritis (obliteration of the margin of the disc, great engorgement of veins, with sometimes hæmorrhages here and there), and a diffuse white coloration over a large portion of the fundus.

The appearance of such a tubercular mass in the choroid would probably not be difficult to diagnose if attention were paid to certain points which Horner has shown distinguish it from sarcoma on the one hand and purulent choroiditis on the other—the two affections which it most closely resembles. From sarcoma it may be distinguished by the flatness of the swelling when compared with its superficial extent, by its more or less distinctly nodular form, and by the evident inflammatory changes in the surrounding choroid. Again, it differs from purulent choroiditis in there being no history of injury or any condition likely to set up that form of inflammation, as well as by its being relatively more circumscribed and localised to one portion of the choroid.

Miliary Tubercle of the Choroid is only met with in cases where there is already, or, much less frequently, where there is afterwards an infiltration of the same nature in a number of other organs. It is probably never met with where tubercles exist in the lungs alone, and it appears to have no definite relation to tubercular meningitis. In fact it is if anything least common in children in whom tubercular meningitis is most common. "It seems as if," as Horner remarks, "the pia cerebri takes the place of the pia oculi." Of thirty-nine cases of tubercle of the choroid, for instance, examined by Litten, tubercular inflammation of the brain or its membranes was only present in nineteen. Litten's thirty-nine cases occurred in fifty-two cases of general tuberculosis. As the tubercles are late of appearing in the choroid, or at all events of becoming visible on ophthalmoscopic examination, it is seldom that the diagnosis of tuberculosis is facilitated by the use of the ophthalmoscope. When present they afford a positive proof of the tubercular nature of the general disease, while their absence does not exclude the possibility of it. It appears certain that choroidal tubercles occur at some time or other in by far the greatest proportion of all cases of miliary tuberculosis, but they probably do not invariably appear, as was at one time supposed, on the authority of Cohnheim, to be the case.

A considerably larger number of tubercles is always found on post-mortem examinations than can be seen with the ophthalmoscope. This is due partly to some being located in that part of the choroid which cannot be seen, but mainly to their not all having advanced so far as to destroy the pigment layer in front of them. When making their appearance, as they often do, only a few days or hours before death, the state of the patient may be such as to render an ophthalmoscopic examination difficult.

Miliary tubercles generally occur in both eyes, sometimes only in one, and always in the choroid,—never in the retina. Their number varies from one to ten or more, though most frequently three to six seems to be the number seen. They are always round, and vary in size from one-sixth the diameter of the papilla, or less, to quite half as big again as the papilla. Usually the size in any particular case is from one-third to two-thirds the diameter of the papilla. The larger ones are distinctly prominent. Their colour is very distinctive, and makes it all but impossible to confound them with any other form of patch characteristic of the numerous varieties of disseminated choroiditis. The regular transition from the normal red colour of the fundus at their margins to the dull yellowish-white at their centre, according as the overlying pigment is more or less completely pushed aside, was first described by von Graefe and Leber.

The following description is taken from Horner, whose experience of such cases was unusually large :—

"The centre is a dull white, and this is surrounded by a dull rose-coloured area; even in that part which exhibits no prominence quite at its margin the red colour of the fundus has still a dim appearance (behaucht). Nowhere is there the sheen and the bluish tendinous discoloration of exposed sclera, or the bluish-white colour of small detachments of the retina, caused by acute exudations or large hæmorrhages. Inflammatory patches of fresh disseminated choroiditis may also be readily distinguished from them. When these patches are of quite recent date, the thinning of the pigment which they occasion causes them to assume the appearance of flat irregularly-shaped spots of a brown and yellowish-red colour. The diagnosis of older patches is rendered certain by the exposure of larger choroidal vessels, and of the sclera as well as the pigmentation of the margin. In this case, too, the margins are much more sharply defined than in the choroidal tubercle, which is always surrounded by a narrow ring of slight opacity where it merges into the saturated red of the fundus. The absence of any nodular prominence readily demonstrated in the larger tubercules, especially where a retinal vessel crosses it, is of importance in the connection with the differential diagnosis."

Miliary tubercles in the choroid give rise to no pain, and apparently to no interference with vision. Anatomically they are like the same structures elsewhere, composed of lymphoid cells, amongst which are giant cells and fattily degenerated detritus.

HÆMORRHAGES IN THE CHOROID.—Hæmorrhages are hardly of such frequent occurrence in the choroid as in the retina. The smaller choroidal hæmorrhages may usually be distinguished from the retinal ones by their not having any constant relation to retinal vessels, and not presenting the flame-shaped appearance which characterises retinal hæmorrhages in the nerve fibre layer. When near the retinal vessels, these vessels may sometimes be seen to pass over them. Large choroidal hæmorrhages again are not bordered by such a sharp and regular line as is the case in large retinal extravasations. It is impossible, however, with certainty to diagnose the position of a hæmorrhage, as those originating in the deep layers of the retina, when of moderate size, exactly resemble hæmorrhages in the choroid. There is more tendency, possibly owing to the resistance offered by the lamina vitrea, for a choroidal hæmorrhage to pass backwards than into the retina. When large, the choroid may be detached by the accumulation of blood between it and the sclera.

Choroidal hæmorrhage may be of traumatic or idiopathic origin, and, according to its extent and the position of the extravasation, may interfere greatly or not at all with sight. On absorption it leaves an atrophic patch, bordered with pigment.

The presence of hæmorrhages, when idiopathic, should direct attention to the general health, and the *treatment* be regulated according to the result of such an examination. Diseases of the blood and the blood-vessels are the cause, as in the case of the retina, of choroidal apoplexy. In all cases perfect rest should be given to the eyes, by avoidance of strong light, or of any occupation necessitating vision near at hand.

OSSIFICATION OF THE CHOROID.—In eyes which have been lost by irido-cyclitis, accompanied by an exudative choroiditis, true bone may in the course of time be found in the choroid. Only the inner layers, chorio-capillaris, &c., of the membrane, are as a rule involved, and it appears certain that the bone forms in the exuded matter, and not in the original tissues of the choroid. The process of bone-formation in this situation has been studied by several observers, and all stages of ossification have been met with. It does not in any way differ from the development which takes place from periosteum. The plate of bone formed takes the shape of the inner surface of the eye. The formation always ceases at the border of the ciliary body in which ossification never takes place. There is therefore evidently some intimate connection between this pathological structure and the chorio-capillaris. I have always seen an aperture left at the position of entrance of the optic nerve, but it is said sometimes to be bridged over. Frequently calcareous degeneration is found in the cataractous lens at the same time.

As the eyes in which ossification takes place are otherwise destroyed, the *diagnosis* can only be made by feeling a hard body, which ends sharply some 4 or 5 millimetres from the corneo-scleral margin.

A probably altogether unique case is recorded by Laqueur, where, after the extraction of a dislocated calcareous lens, the media were found to be sufficiently clear to admit of ophthalmoscopic examination. In this case no trace could be seen of the optic nerve or retinal vessels, and the appearance altogether seems to have been very remarkable, while the diagnosis of ossification of the choroid was confirmed by palpation.

The presence of bone in the choroid is not of itself sufficient to give rise to sympathetic inflammation, but cases are on record in which this result has followed an irido-cyclitis evidently set up by irritation from the ossified spicula. The *treatment* should therefore be to remove such eyes, as they may at any time be the cause of serious mischief.

Detachment of the Choroid.—The choroid may be detached from the sclera either traumatically or idiopathically, and by blood, by a serous effusion, or by tumour. Detachment of the choroid is very uncommon, or at all events it is not frequently diagnosed. I do not know that there are any signs which would enable one at once to diagnose this condition from ophthalmoscopic examination alone. Cases in which detachment of the choroid takes place from any cause, appear generally to end in shrinking of the globe.

I have only once met with a case of simple detachment of the choroid, and it was only after repeated examination that the diagnosis could be made. The appearance was exactly that presented by a tumour situated below and detaching the retina, but the tension was low, and no change took place, except further reduction of tension during six months. The detachment stretched out to the middle of the vitreous chamber, and came in the way of the direct line of vision, so that no central fixation was possible. There was no tremulousness of the detachment, such as there would certainly have been had the retina alone been detached to that extent, and there was an absence of transparency.

While, therefore, the differential diagnosis between simple detachment of the retina alone, and detachment of both retina and choroid, could be made with tolerable certainty at once, the course of the case had to be watched before the other condition liable to be confounded with detachment of the choroid, viz., tumour growth behind the retina, could be excluded.

DISEASES OF THE VITREOUS.

Acute purulent inflammation of the vitreous may occur when a foreign body is lodged in it, or it may be the result of the extension of some inflammation from other parts of the eye. The question as to whether the inflammatory products in the first case are derived from alterations in the elements of the vitreous itself, or have migrated from the vessels in the surrounding coats towards the focus of irritation, is one which has been much disputed, and which can hardly yet be said to have been definitely settled.

From a clinical point of view the true pathology of *purulent hyalitis*, as it is called, is a matter of little importance. When once it is set up, the eye is doomed, and eventually shrinks. This result may or may not be preceded by perforation with evacuation of more or less pus. Less severe inflammatory conditions of the vitreous probably never occur primarily, but are always associated with inflammation of the ciliary body, choroid, or retina. Clinically, however, the condition of the vitreous is often the point of primary importance, as the passage of inflammatory exudation into its substance gives rise to opacities on the existence of which the defect of vision accompanying the particular inflammation may mainly or entirely depend. This is, for instance, the case in cyclitis, and to some extent also in syphilitic choroiditis.

Opacities of the Vitreous may be stationary or floating. They may be diffuse and punctiform, filamentous, flaky, or membranous. These opacities are mainly of two kinds, either due, as has already been said, to products of inflammatory exudation from the vascular membranes of the eye, or to the effusion of blood from the vessels of these membranes. It seems probable, though it is by no means certain, that some, too, are caused by coagulations of some of the elements of the vitreous itself. As the result of exudation into the vitreous, the gelatinous consistency which it normally presents may become altered. The vitreous may thus become abnormally liquid or abnormally condensed and shrunken.

Liquefaction of the vitreous, or *synchisis*, may be diagnosed with certainty when that body contains more or less organised opacities which alter their position with great rapidity with the movements of the eye. There is often at the same time increased intraocular tension. All cases of liquefaction of the vitreous are not accompanied by floating opacities. Thus we may see cases where the lens has been dislocated into the vitreous, and in which, from its free movement up and down with the movements of the eye, a tolerably complete liquefaction of the vitreous may be inferred to have taken place, in which the transparency is complete.

The diagnosis of liquefaction without opacity may be extremely difficult. The cause of such cases, too, is often very far from being apparent. Sometimes it appears to be a senile change unconnected with any choroidal disease. Condensation of the vitreous, on the other hand, may generally be inferred when, along with more or less diffuse and stationary opacity, there is considerable diminution in the tension of the eye. This condition is likely sooner or later to be complicated by detachment of the retina.

Diffuse opacity of the vitreous is a most constant accompaniment of syphilitic choroiditis. On careful ophthalmoscopic examination by the direct method, the opacity which at first sight appears to be diffused, and to veil the image received of the back of the eye, may be seen to be punctiform, or made up of a number of closely packed dots. It will be seen, too, not to occupy the whole vitreous, but to be confined to certain layers, so that to be properly seen certain distances must generally be focussed for. When this is done, the opacities will often be seen to sway about, so that they are in reality hardly as stationary as might be supposed on superficial examination. They cause, as a rule, greater visual disturbance than circumscribed floating opacities, the patient complaining of a cloudiness over the objects seen, and often at the same time describing this veiled appearance as swaying to and fro. Such diffuse opacities frequently partly clear away, and then reappear several times during the course of the disease. They usually last for months, and eventually, as a rule, entirely disappear.

Exudations or effusions sometimes coagulate into filamentous or thread-like opacities, which, when freely moveable, are projected by the patient as snake-like black objects, which are continually changing their shape and position. No doubt such opacities have given rise to most of the stories of thread-worms in the eye. Membranous opacities are either free and capable of assuming as they fold or unfold numberless forms, or they are fixed to the papilla or retina and only sway about at their free ends. Sometimes these fixed membranes contain newly formed blood-vessels, but the vascularisation of vitreous opacities is rare. A few cases have been recorded where a fine membranous network containing blood-vessels has been seen in the vitreous chamber, apparently the remains of a fœtal condition.

Prolapsed portions of vitreous often become rapidly vascularised, but this does not extend in depth, and on the whole there is little tendency to opacity following accidents leading to loss of vitreous, provided they are not followed by inflammation of the margins of the wound in the ocular coats.

Bleeding into the vitreous takes place owing to the giving way of some vessel or vessels in the retina, choroid, or ciliary processes. This may be the result of a trauma or be due to a diseased state of the vessels or of the blood. When the patient observes the pretty sudden defect of vision which such a bleeding may occasion, the cloud or veil which comes in front of his sight often appears at the same time of a distinctly red colour. When the extravasation is very large, the blindness produced is pretty complete. Most commonly, however, only a portion of the vitreous is infiltrated with blood, and as this gradually gravitates to the most dependent part, the cloud is generally thickest in the upper portion of the field of vision. Great differences are observed in the rate of absorption of the blood, differences which depend partly upon the age of the patient, and partly, no doubt, also upon the state of the choroid and retina. Often a particularly slow absorption is really due to the recurrence every now and then of hæmorrhages before a previous extravasation has properly cleared away. The absorption may be complete, or may leave flaky or irregularly shaped opacities, which alter their position with the movements of the eye with more or less rapidity, according to the fluidity of the vitreous. These masses are projected as black spots or patches, which appear to be constantly in motion when the eye is moved, and to fly or move slowly upwards when it has come to rest.

When the normal consistency of the vitreous has not been much interfered with, the blood-clot may keep pretty much in the line of sight, and thus greatly interfere with vision; or the patient may learn, by making a sudden movement of the eye, to get rid of the dense cloud for a few seconds, and thus be able to read, but he has constantly to repeat this movement, and is only able to make out a few words, it may be, at a time. This symptom of only being able to read a few words at a time, without suddenly altering the direction of fixation, is indeed, as von Graefe first pointed out, quite characteristic of a large blood-clot in the vitreous.

To ophthalmoscopic examination the blood lying in the vitreous appears red if fresh, and black if some time has elapsed since the extravasation took place. Often the red reflex from the surface of the blood-clot may be made out by oblique illumination, if the eye be brought as nearly as possible in a line with the convex lens. It appears to be more commonly the choroid than the retina from which extravasations of blood take place into the vitreous. At all events, subsequent changes are most frequently found in that coat. Bleeding, too, often takes place from the anterior portions more readily than from the more central, a circumstance which is of importance in so far as the vision is on this account less likely to be permanently damaged.

A somewhat rare but distinct clinical type of disease is the occurrence of repeated hæmorrhage into the vitreous in young individuals. This affection was first described by von Graefe in 1854, and has since hardly received the attention it deserves. It seems to result from periodic capillary hyperæmia of the choroid, and in some way to be associated with irritation in the generative organs. It is most common in young men of effeminate, as distinguished from active or energetic temperament.

In the cases which have been observed in girls, the connection with menstrual disorder has been most marked. Thus Dor has recorded a case where the tendency to repeated hæmorrhage into the vitreous ceased in a young girl as soon as menstruation had become established. Epistaxis is a common accompaniment, or rather is met with at other times in cases in which there is this tendency to hæmorrhage into the eye. As there is never found to be any diathesis which might account for an altered condition of the blood, it seems probable that the true pathology is more of the nature of a neurosis. On this point Eales, who has observed a number of cases, remarks, "from the character of the hæmorrhage, and from the evidence of local variations of circulation, and from the slow pulse, constipation, flushing of the face, headache, and puffiness and discoloration of the eyes, I am inclined to attribute this combination of conditions to a neurosis affecting both the circulatory organs and the digestive system, leading on the one hand to partial inhibition of the muscular movements of the bowels, and to a vasomotor contraction of the vessels of the alimentary canal, with inhibition of its secretory functions, thereby causing dyspepsia, constipation, malnutrition; and on the other hand, to a compensatory dilatation of the systemic capillaries, especially those of the head, and in these cases of the retina causing over-distension of the venous system and systemic capillaries, with liability to rupture on the occurrence of any intensifying cause. Hence the headache, the epistaxis, the retinal hæmorrhages, and the tortuosity and fulness of the retinal vessels and temporal artery." Often both eyes may be affected, but probably rarely if ever at the same time. There seems to be no greater tendency to the bleeding taking place in the one eye than in the other, though in the cases recorded by Eales the left was most frequently affected. It seems probable, too, from his cases that the hæmorrhage may sometimes come from the retina. This view is also entertained by Leber, or was at all events in 1876, at the time of publication of his great work on the diseases of the retina. He gives, indeed, a drawing of such retinal hæmorrhage in a case observed by Weber. The ophthalmoscopic appearances met with in most cases, however, on the clearing away of the blood in the vitreous, which have been well described by Nieden, leave no doubt that the extravasation usually takes place from the choroid. It is remarkable, indeed, how often one sees retinal hæmorrhages without any extravasation into the vitreous. It would appear that the cases in which the origin of the hæmorrhage is retinal are likely to go on to glaucoma.

In some cases recovery is eventually complete, though when there have been repeated attacks this is not generally the case. It appears possible that some may proceed to the formation of connective tissue bands on the retina, and thus be the beginning of retinitis proliferans.

A peculiar form of vitreous opacity, which gives rise to a very beautiful ophthalmoscopic appearance, is that caused by the accumulation of cholesterine and other crystals in the vitreous. As at the same time the vitreous is liquid, this condition has received the name of *synchisis scintillans*, from the glistening caused by the reflection of light from the surfaces of the crystals. On ophthalmoscopic examination the glittering spots, which have much the appearance of small particles of gold suspended in a fluid, are seen to dance about with the slightest movement of the eye.

Whether these crystals are the result of fatty and other degenerative changes which take place in the elements of the vitreous itself, or of similar changes in substances which have passed into it from the choroid, does not seem very clear. The first origin is not improbable, from the fact that often no other changes are to be seen in the eye, and the vision is not much reduced.

The condition is one met with almost exclusively in old people. When found in younger eyes it is usually, if not invariably, secondary to some serious alterations which have very much impaired the sight. It often remains stationary for years, and does not admit of, or indeed call for, any treatment.

The *prognosis* in cases of vitreous opacity depends on the extent to which the organisation of the opacities has gone. When the eye is otherwise healthy, even opacities which have lasted for several months may eventually disappear. When the vitreous is very liquid, especially if, at the same time, there are extensive choroidal changes, the condition is likely to remain stationary, or at all events not to improve.

The *treatment* has mainly to be directed against the cause of the opacities, so far as that can be discovered. The constant electrical current has been said to promote absorption even of floating opacities. This treatment does not seem to have been much employed, and is no doubt utterly without effect. Other more trustworthy methods of promoting absorption should be tried, such as pilocarpine injections, wet packing, &c.

PERSISTENT HYALOID ARTERY.—In the embryo an artery passes from the central artery of the retina to the back of the lens, where it divides into a network of vessels destined for the nourishment of the lens. This disappears altogether before the end of fœtal life, but the transparent sheath by which it is surrounded persists, and forms the so-called central canal which was discovered by Stilling. This canal is probably of some importance, either in connection with the nourishment of the lens or the removal of effete products from the vitreous chamber. Very rarely more or less pronounced rudiments of the artery remain. In such cases an opaque, usually somewhat tortuous, cord can be seen with the ophthalmoscope to stretch from the centre of the papilla forwards to the back of the lens. Sometimes there is at the same time some other persistence of a fœtal structure, such as remains of the pupillary membrane, &c., and the vision is generally not of normal acuity.

CYSTICERCUS IN THE VITREOUS.—The presence of a cysticercus in the eye is altogether an extremely rare occurrence in this country. The disease is met with tolerably frequently in North Germany; in Berlin, for instance, according to some statistics, once in about every five hundred cases treated in the ophthalmic hospitals. Its development takes place between the retina and the choroid, and gives rise to detachment of the retina. At this stage it is almost always possible from the colour and shape to diagnose the cause of the detachment. Later on perforation of the retina takes place, and the parasite passes into the vitreous. So long as the vitreous retains its transparency the vesicle can be distinctly seen, and its nature diagnosed by the movements of the neck, which at times is pushed out and retracted. Indeed, the appearance of a round and freely movable vesicle is of itself sufficiently distinctive. The vitreous becomes sooner or later turbid owing to the setting up of irido-cyclitis, and the diagnosis may thus be rendered difficult or impossible. If left alone the inflammatory changes to which it gives rise lead to destruction of the eye.

The *treatment* therefore consists in attempting to remove the cysticercus. Such operations have been frequently performed, and with very great success. Vision is not always preserved, but in most cases, at all events, the eye comes to rest without shrinking, and without the necessity for enucleation. The best chances are presented by cases in which the vesicle is still sub-retinal. The success of an operation depends greatly on the correctness with which the position occupied by the cysticercus can be localised. When at the back of the eye it suffices, according to Alfred Graefe, to determine how many diameters of a papilla the vesicle lies from the disc. In other situations he makes use of a specially constructed localising ophthalmoscope, which enables him to calculate with sufficient accuracy its distance from the corneal margin. Of forty-five cases operated upon by him, twenty were completely successful. One case is recorded by Hansen Grut, in which extraction of the parasite from the vitreous was followed by the retention of full normal visual acuity. Two different cysticerci have occasionally been met with in the same eye, but there appears to be no record of both eyes ever having been affected.

DETACHMENT OF THE VITREOUS.—The vitreous may be detached from its connection with the retina by a blow, or as the result of exudation taking place slowly, as in cases of sclero-choroiditis posterior. The space left is then permanently occupied by fluid in which there may or may not be small floating opacities. The condition is no doubt frequent in the higher degrees of myopia with bad vision, also as a forerunner of many, if not all, cases of detachment of the retina; but there do not appear to be any constant signs by which it can be diagnosed with certainty.

CHAPTER IX.

FOREIGN BODIES IN THE EYE.

Foreign Bodies in the Anterior Chamber.—A foreign body may enter the anterior chamber by passing directly through the cornea, and this is much the more common way; or it may reach this position after penetrating through the iris or lens, entering the eye through the sclera. The wound in the cornea is generally in its lower half, as the upper is mostly covered by the lid, but it sometimes happens that the lid is perforated before the cornea. In that case, however, the force of propulsion mostly carries the body deeper into the eye, lodging it in the vitreous, or choroid, or sclera, or even in the orbit beyond.

The presence of a foreign body in the anterior chamber may cause more or less severe inflammation of the iris and adjacent parts, leading in the worst cases, if not removed, to eventual destruction of the eye. The inflammation, on the other hand, after continuing for some time may subside, and the foreign body become encapsuled. In some cases it happens that the foreign body lies free in the anterior chamber, without producing any irritation, or it may become absorbed, with or without at the same time setting up inflammatory reaction. The first result is by far the more common. It occasionally happens that the inflammation produced is limited to the anterior portion of the eye, and this may eventually lead to perforation and expulsion of the foreign body through the cornea. More frequently, however, it causes either slow insidious irido-cyclitis of the worst type, or more active purulent irido-choroiditis, which is followed by phthisis bulbi. Encapsuling is very rare, but it not so infrequently happens that no irritation is produced at all, and this result is most common when the foreign body is a piece of coal or carbon, or of some chemically inactive metal.

No doubt the effect produced will depend in great measure upon whether or not septic matter has been introduced into the eye at the same time. It is not easy in any given case to make sure of this point, but as a general rule hot pieces of metal are aseptic, and accidents with such pieces admit of a better prognosis from the first. Something depends on the nature of the metal itself.

Thus Leber has found, by introducing aseptic portions of metal into the anterior chamber of rabbits, that copper gives rise to much more irritation than steel or iron. Aseptic portions of a sewing-needle introduced into the anterior chamber of a rabbit produced the following changes :—Slight injection round the lower border of the cornea, followed after a few days by a thin scale of yellowish exudation round the metal; no opacity of the aqueous humour or exudation beyond the immediate proximity of the foreign body. If the point of the needle came to rest against the cornea, it gradually made its way through it. The effect produced by similar portions of copper was very different. If they touched the iris, they produced a marked ciliary injection and great hyperæmia of the iris, followed by a copious purulent deposit round the metal. The cornea in the vicinity of the metal became more or less infiltrated, and sometimes totally destroyed, whereupon expulsion took place, and a relatively good cure might result. The inflammation was always, however, confined to the anterior part of the eye, *i.e.*, to the neighbourhood of the foreign body. In another series of experiments Leber introduced into the anterior chamber a number of fine particles of copper or iron. In the case of copper, each particle became surrounded by a purulent deposit; after some time the inflammation subsided, and the particles of copper disappeared without leaving a trace. In the case of the iron particles, absorption also took place, but was accompanied by much less irritation. A piece of copper wire pushed through the cornea into the lens, and left with the free end in the aqueous chamber, was found to produce hardly any irritation at all, owing, as Leber believes, to the dissolved particles of copper being at once taken up by the surrounding albumen before coming in contact with the vascular portions of the eye.

FOREIGN BODIES IN THE IRIS.—The iris is on the whole a rather uncommon site for a foreign body within the eye, as the force which propels it so far is mostly sufficient to carry it further. The presence of a foreign body in the iris is likely to lead to much the same results as those just described when it lies in the anterior chamber. Absorption is less likely, and encapsuling more likely. I have, for instance, seen a case where a piece of steel remained encapsuled in the iris for fifteen years without producing any irritation.

The first effect which a foreign body produces in the iris is to give rise to hæmorrhage, which in most cases is so slight as to already have become absorbed, or to have left but faint traces, by the time the case comes under observation. Most frequently this is followed by iritis, the symptoms of which do not become prominent until a day or two after the accident. According to the size and nature of the foreign body, and the presence or absence of micro-organisms, the iritis, which its presence produces, may be purulent, or merely plastic. In the first case, the complete destruction of the eye takes place rapidly by panophthalmitis; in the second, the foreign body may become encapsuled, or inflammation may uninterruptedly, or by recurrent attacks, cause loss of vision in the way commonly met with in cases of old-standing iritis.

FOREIGN BODIES IN THE POSTERIOR AQUEOUS CHAMBER.—Foreign bodies usually lodge in the posterior aqueous chamber by penetrating the eye slantingly, at or near the corneo-scleral junction. Sometimes the external wound is found to be in the central portion of the cornea. A foreign body in this position almost invariably, and especially if it be of any size, sets up inflammation in the iris and ciliary body, and often gives rise at the same time to cataract.

The Diagnosis of Foreign Bodies in the Aqueous Chambers and Iris.—The history of the accident must be carefully inquired into. If it turns out that the eye has been forcibly struck by a small body, there is the possibility or probability that it has penetrated and remained in the eye. The cornea should then be examined by oblique illumination for any appearance of a scar. If none be found on the cornea, the sclera should also be examined, but the absence of any visible cicatrix there is not conclusive evidence against the previous existence of a wound. Fortunately for the diagnosis the wound produced by a foreign body lying in the anterior section of the eye is at least nine times out of ten in the cornea.

When a fresh wound or scar is discovered in the cornea, the question arises, has or has there not been perforation? To determine this we must observe—the shape of the pupil, the depth and contents of the anterior chamber, the condition of the iris and lens, and the tension of the eye. We must also notice whether any synechiæ, anterior or posterior, have formed, or whether there be any prolapse of iris or vitreous, or any trace of uveal pigment in the corneal wound. It is only in the case of relatively large foreign bodies that the tension is reduced or the chamber shallowed, unless the patient happens to be seen immediately after the accident, when the diagnosis of the perforation is easier. Any prolapse is of course a certain evidence of perforation, while wounds of the iris or lens, hyphæma, hypopyon, and synechiæ, point more or less conclusively to the same.

When taken along with the history of the accident having been caused by a small body, any or several of these evidences of perforation render it extremely probable that a foreign body is lodged in the eye, and the next point is to determine where. In the first place, is it in the anterior or posterior section? The most important aid towards the solution of this point is afforded by the examination of the vision and field of vision. In all cases where the anterior portion of the eye is the site of the foreign body, the vision corresponds to the visible optical changes, whereas this is not the case where the posterior portion of the eye is that into which it has penetrated. If it be in the posterior aqueous chamber, the diagnosis is always difficult, as it is always hidden by the iris, unless, as sometimes happens, a portion of it may be sticking out beyond the pupillary margin, where it may be seen if the aqueous and cornea be clear. When in this position, it often happens, too, that the iris has been penetrated, and the diagnosis thus facilitated. The presence of a foreign body in the posterior chamber, if it be not very small, is always indicated by a protrusion of the iris over it, or a drawing up of the pupil towards the spot. Sometimes these appearances are so slight as to make the supposed cause very uncertain, and indeed, it occasionally happens that the diagnosis of the presence of a foreign body in this position is impossible.

Although the anterior chamber and iris are freely exposed to view, the existence of a foreign body in either position is not always easily diagnosed. When in the anterior chamber, it may have sunk down into the angle, and if small, be difficult of detection; or the cornea may be hazy at its periphery, or the chamber occupied by more or less pus or blood. When of any size, if the cornea is clear, and there is no hyphæma or hypopyon, the body can generally be seen, either, in fresh cases, in its natural state, or in cases where some time has elapsed since the accident, surrounded by lymph, appearing then usually as a sharply defined yellowish mass. When not thus directly visible, the points which nevertheless render the diagnosis of its presence more or less certain, are a localised circumcorneal injection, and tenderness to pressure, with some retraction of the pupil in a particular direction, usually downwards, where the foreign body commonly lies. Apart from this, the existence of inflammatory reaction, the severity of which would be greatly out of proportion to the trauma if no foreign element existed in the eye, is of very considerable diagnostic importance.

If in the iris, a foreign body can generally be seen, though, if small, some care is necessary in the examination, and a doubt may sometimes exist as to whether a particular appearance is actually caused by the presence of a foreign body, or is merely a pigment spot. Sometimes, when the iris is prolapsed, the foreign body is found in the prolapsed portion.

Treatment.—Very small portions of iron or steel, if they have caused little irritation, may be left, on the chance of absorption taking place in the course of time, but the patient should be kept constantly under observation, so that the first symptoms of irritation, should they afterwards arise, may be noticed, and an attempt be made to remove the body. In all cases of iron or steel, the simplest method of removal is with the electro-magnet. The incision necessary for their removal should be in the corneo-scleral margin, and except when the piece of metal is sticking in the iris, it should be made at the lower part of the cornea. The size of the incision must be regulated according to the size of the foreign body to be removed, and should always be bigger than it is, so as to prevent its being rubbed off as the magnetised probe is withdrawn. Portions of the non-magnetisable metals or other substances have to be removed with a curette, or pair of *ribbed* (not fixation) forceps. In the case of small bodies lying in the iris, the curette is usually the simplest; for larger objects, especially when they lie free in the anterior chamber, the forceps are preferable. In any case, the wound at the corneo-scleral margin must be made of sufficient size. Should it be found, after the foreign body has been seized, that it cannot be withdrawn, and sometimes it turns out to be a little bigger than it appeared to be, the operator must *not* let go his hold of it, but proceed as best he can to enlarge the wound until it admits of its removal. Where any doubt exists in the diagnosis, a paracentesis made with antiseptic precautions, so as to admit of the escape of the fluid contents of the anterior chamber, should be undertaken.

In the case of foreign bodies embedded in the iris, it is usually necessary to excise the portion of iris in which they lie as well, but an attempt may be made in the first place to remove the foreign body alone. If this can be done without much laceration the iris may be left; if not, it should be excised. In one case, where I removed a piece of steel 4 mm. in length from the iris, the operation was not followed by even a trace of iritis.

Foreign bodies in the posterior chamber are usually difficult to extract; almost invariably it is necessary to remove a portion of iris as well,—sometimes the lens has also to be extracted. The best plan is to attempt to seize the foreign body through the iris, and cut off all that is withdrawn through the opening made for the removal. When the iris is prolapsed in an accident which renders the lodging of a foreign body in the eye possible, it should be carefully examined and bathed with an antiseptic lotion before being returned into the eye. If it is much bruised, or some time has elapsed since the accident, it is better to cut off the prolapsed portion.

All operations performed for the removal of foreign bodies require great steadiness on the part of the patient, and, unless this can be insured, should not be undertaken without an anæsthetic. For such cases, in children at all events, cocaïne is insufficient. How soon and how completely the irritation produced by the presence of the foreign body will subside, depends greatly on its size and nature, and the length of time it has remained in the eye. The possibility of sympathetic inflammation of the other eye has always to be taken into consideration in the treatment.

FOREIGN BODIES IN THE LENS.—When a foreign body enters the eye at all, it is perhaps on the whole most fortunate for the patient if it happen to lodge in the lens. Little or no inflammatory reaction follows, and there is, practically speaking, no chance of sympathetic ophthalmitis. Inflammation may arise owing to the wounds produced by the perforation of other superjacent structures, principally the iris and ciliary body.

A small foreign body passing through the centre of the cornea, and lodging in the lens without traversing the iris, may produce so little irritation or disturbance as to leave the patient ignorant of having sustained so severe an injury, until the vision becomes gradually more and more impaired by the advancing opacity of the lens. A foreign body in the lens, in fact, almost invariably causes cataract. In rare instances only a limited opacity surrounding the foreign body results, as, for instance, in the case of small particles of gunpowder. The cataract is no doubt frequently caused more by the imbibition of aqueous humour through the rupture in the capsule than by the actual presence within the lens of the foreign body, and great differences are consequently met with in the rapidity with which the cataractous change advances—differences depending on the size of the opening in the capsule, and the length of time which it remains open, as well as on the age of the patient. Small capsular wounds, caused by small bodies, heal of themselves; larger ones are often plastered over by the over-lying iris.

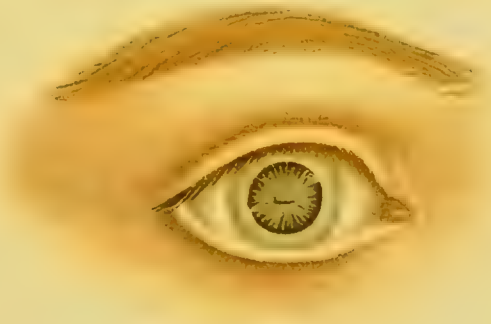

FIG. 93.—Piece of steel in lens.

A lens which becomes cataractous by lodging a foreign body is eventually partially, and in some rare instances completely or almost completely, absorbed, and the foreign body may, after a longer or shorter period, become displaced into such a position as to give rise to serious inflammatory disturbance. More or less irritation is always caused when the capsular wound is sufficiently large to allow of the escape of lens matter into the anterior chamber. The swelling of the lens within its capsule is liable at the same time to take place. These accidents are better tolerated in the young than in older individuals.

Only very rarely do small foreign bodies lodge in the lens capsule. They cause neither much irritation nor cataract, only a slight opacity in the capsule itself.

Diagnosis.—When penetration has taken place through the sclera, the chances of the foreign body being in the lens are very small, but it may have passed in obliquely, either through the ciliary body or from the posterior aqueous chamber. When evidence of a perforating wound has been found in the cornea, with the history of a small particle of metal or other substance having been propelled against the eye, and yet no appearance of a foreign body in the chamber or iris, the capsule of the lens should be carefully examined by oblique illumination, and with the pupil well dilated by atropine. In difficult cases, examination of the capsule may be preferably made with the ophthalmoscope, by reflecting light into the eye, and bringing a strong convex lens in front of the aperture. In this way the wound or scar in the capsule will almost always be detected. When this is discovered it is certain that the foreign body has entered the lens, and the point remaining to be determined is, whether it has passed through it or lodged within it. The diagnostic evidence in this respect is either positive or negative. In the first place, in a good many cases it may actually be seen on oblique illumination or on examination with the ophthalmoscope, in the manner described. If metal, it then presents a glistening appearance. Sometimes, though the foreign body may not be directly visible, an area of greater saturation may be made out in one particular part of the lens, occasionally with a distinct streak of opacity running up to it. This portion, or it may be the whole lens, may at the same time exhibit more or less coloration, produced by oxidation or otherwise. The negative symptoms of importance have mainly to be relied on when the whole lens has become opaque. But even where there is considerable transparency, the diagnosis will be strengthened by finding no evidence, on ophthalmoscopic examination, of the presence of any body in the deeper parts of the eye. The negative evidence in favour of the lens being the site of the foreign body known to have entered the eye, consists in the first place in a disproportion between the lesion and the irritation set up by it, and also in the absence of positive moveable scotomata or of restrictions in the field of vision, such as are indicative of the presence of foreign bodies in the vitreous.

The *treatment* will depend on the degree of opacity to which the foreign body has given rise. In most cases it is inadmissible to perform any operation until complete cataract has resulted. The exceptions to this are cases where a glaucomatous condition is set up by the swelling of the lens in its capsule, and in which, therefore, immediate extraction is indicated. In other cases, too, in which the opacity in the lens is incomplete and practically stationary, operative interference seems sometimes justified before a spontaneous ripeness has been arrived at. In such cases some method of artificial ripening may sometimes be resorted to first. It is rarely advisable, in any case where a foreign body is lodged in the lens, to attempt any operation but the removal of the cataract through a large section. When the body is a magnetisable metal this may be done in the ordinary way by first rupturing the capsule with a cystotome. The piece of metal then usually comes out along with the cataractous lens, but should it remain behind, it is generally possible to extract it with the electro-magnet, which should always in such operations be at hand. In cases where the piece of steel or iron is seen lying immediately behind the capsule, the magnet should be used before completing the extraction. In the case of non-magnetisable substances, although they may often in the same way be removed along with the cataract, it is safer on the whole to extract the lens in its capsule, if the case admits otherwise of the performance of this operation.

FOREIGN BODIES IN THE POSTERIOR SECTION OF THE EYE.—Considerably the largest proportion of cases of foreign bodies in the eye are those in which the body has penetrated into the deeper structures. This is unfortunate, because of the uncertainty of our being able to remove the foreign body, and because of the tendency to sympathetic inflammation.

Large portions of metal always produce so much destruction by their mere presence, that the case may be hopeless from the first, while in all but a very few cases the chances of operative interference being successful will depend almost entirely on whether the patient is seen soon after the accident or not. Very frequently they present themselves too late.

As in the case of foreign bodies lodging in the anterior portions of the eye, those which are found in the vitreous and posterior coats pass into the eye, most frequently through the cornea, wounding the iris and lens, or the lens alone, on their way. Some, and more particularly shot pellets, may first traverse the sclera and then enter the vitreous chamber. A body propelled in this way through the sclera may, according to the inertia it has, be arrested in the coats of the eye, at the same side of and near the aperture of its entrance, or it may drop into the vitreous, lodge in the coats of the eye at the opposite side, or penetrate these and come to rest in the tissues of the orbit. This last occurrence is not so very uncommon in the case of gunshot injuries. If the foreign body does not remain permanently fixed in the coats of the eye at either side, it sinks sooner or later in the vitreous, and then only alters its position owing to contractions which may take place in the exudation surrounding it.

The first effect produced as a rule by a foreign body entering the back portion of the eye is hæmorrhage from the choroidal vessels. The blood usually accumulates at the anterior part of the vitreous, in which position it may often be made out with the ophthalmoscope. When it lies further back it is suggestive of rupture having taken place near the posterior pole of the eye. When much blood is extravasated it is not long in sinking to the bottom of the vitreous chamber. The vitreous then generally becomes more or less infiltrated with pus, and this infiltration may be accompanied either by violent inflammatory symptoms, panophthalmitis, or these symptoms may be less severe, but eventually also lead to shrinking of the globe. If the course of the case be carefully watched from day to day, the first symptoms of a purulent inflammation are often observed to be linear, in the form of a streak which indicates the passage taken by the foreign body through the vitreous. Not infrequently this streak leads to a place where the infiltration is denser, and it is within this denser portion that the foreign body lies. Occasionally a foreign body, after setting up some irritation, becomes encapsuled in the vitreous; but although the eye then comes to rest, there is always the danger of recurrent attacks of inflammation and ultimate destruction. It should always be remembered that a fresh attack of inflammation, even after there has been a quiescence of years' duration, may give rise to sympathetic ophthalmitis. In cases left to themselves, the greatest security against sympathetic inflammation is afforded by panophthalmitis, particularly if this should happen, as it sometimes does, to lead to the spontaneous expulsion of the foreign body. A protracted or even permanent quiescence is most likely to follow when the foreign body is a shot pellet, for which the prognosis is more favourable than for other bodies in the vitreous.

The result is more favourable when the foreign body, whatever its nature, is situated in the ocular wall. Most frequently it becomes embedded on the same side as that by which it enters the eye, the force of propulsion not being sufficient to carry it into the vitreous. In this position it may cause comparatively little irritation, though more frequently slow inflammatory changes are induced, leading to loss of vision and phthisis bulbi. A localised ulcer may form and the foreign body be spontaneously expelled. When the site occupied is the ciliary body, cyclitis is set up and vision greatly reduced by opacities in the anterior part of the vitreous, followed often by cataract or by more serious changes. When the foreign body becomes lodged in the opposite wall of the eye, which is considerably more uncommon, the chances of its being encapsuled, and good or relatively good sight being retained, seem to be rather greater, though the eye can never, even under the most favourable circumstances, be looked upon as absolutely safe. Inflammation may be set up after long intervals of time. Much depends, in the first instance, no doubt, upon whether or not septic matter is introduced into the eye at the same time. The prognosis is relatively good in the case of shot pellets and small pieces of clean steel or iron.

Diagnosis of Foreign Bodies in the Vitreous and Posterior Coats of the Eye.—The history to be obtained in cases where there appears reason to suspect that a foreign body is lodged somewhere in the back part of the eye is often very unsatisfactory, especially if some time has elapsed since the accident. With an uncertain history, and the absence of any external scar, the diagnosis is often a matter of no little difficulty. The preponderance of cases where the eye is pierced through the cornea, is only about half as great as in the case of foreign bodies in the anterior chamber or iris. According to different statistics, it may be taken roughly that in four out of every five cases the cornea is wounded, instead of nine out of ten. When a corneal wound or scar is found, the diagnosis is of course more easy. Further evidences of perforation are then looked for in the iris and lens. The relative value of such evidence has already been discussed. The vitreous must next be explored with the ophthalmoscope. Owing to the great magnifying power of the eye, it will be possible to see even very small foreign bodies in this situation if they occupy a position which can be got at with the ophthalmoscope, and if the opacities in the lens and vitreous are not too great. It often happens, therefore, that in fresh cases one is able to directly observe the foreign body. By causing the patient to look well down, it will not seldom be found lying at the bottom of the vitreous chamber, and when metallic, will be seen to glisten with a whitish or bluish-white lustre. There is just the possibility of mistaking air bubbles, which are sometimes found in the vitreous after an accident, for portions of metal. Air bubbles are round, often multiple, and glisten at the centre, while they appear dull and obscure at their margins. The glistening from a portion of metal is from its margin, the rest presenting a bluish or greenish-white coloration. If this point be attended to, a mistake or uncertainty cannot arise. When the external wound has been in the sclera it is often not easily discovered, even when fresh, owing to surrounding hæmorrhage or inflammatory hyperæmia. A recent scar may be detected on oblique illumination, and perforation may be inferred when the superficial tissues are tacked down to the deeper, and not freely moveable over them. In fresh scleral wounds the probe may be used carefully, if doubt exists as to perforation.

If the patient cannot furnish satisfactory information with regard to the body by which the eye has been injured, he may possibly remember whether the vision was much affected at the time of the accident or not. Obscuration lasting some days is suggestive of there having been hæmorrhage, more especially if followed by black objects floating in front of the eye, but this is of course not conclusive evidence that anything remains in the eye. When the foreign body, or the yellowish mass of lymph by which it is surrounded, is visible by means of the ophthalmoscope, we may sometimes, if the history is doubtful, as M'Keown recommends, obtain positive evidence of its being steel or iron, by observing a change of position of the whole mass on bringing a powerful magnet in contact with the eye.

The diagnosis of the position of the foreign body when seen by the ophthalmoscope is easily made. When it is lodged in the coats of the eye we find that the same lens is required for accurate focussing of it as for focussing the rest of the fundus, and that there is only very slight or no parallax. Most frequently in such cases there is some evidence of disturbance in the coats surrounding the foreign body, often a whitish patch bordered with pigment, where the choroid has been ruptured at the time of its lodging in the eye. It is only rarely that the glistening of a choroidal plaque may give rise to an appearance so like that of a piece of metal in the eye, that some difficulty may be experienced in making a correct diagnosis. A piece of metal rarely if ever glistens all over, but mainly at its margins, whereas the glistening from a choroidal plaque is not limited to any particular point, and in fact is often all over.

When the foreign body cannot be directly seen, but the history and objective appearances of perforation point to its being present in the eye, a tenderness on pressure at some particular point over the sclera indicates its position in not a few cases. A point, too, of diagnostic importance in a doubtful case is the existence of an amount of inflammation disproportionate to the severity of the trauma alone. This should always give rise to the suspicion that something is lodged in the eye.

Attention must also be paid to the nature of the visual disturbances. Apart from the defect corresponding to the visible optical changes, which alone exist in the case of bodies lodged in the anterior part of the eye, there may usually be made out other defects as well, when the vitreous chamber or coats at the back of the eye are the site of the foreign body. These are of two kinds, positive scotomata and restrictions in the field of vision. The scotoma may be either the projection externally of the shadow thrown on the retina by the foreign body, or it may be due to a lesion in the fundus of the eye. Restriction in the field of vision is due either to the presence of blood or purulent exudation in the vitreous, or to detachment of the retina. This restriction in the field of vision is one of the most valuable points in the diagnosis when it is otherwise difficult on account of opacity of the lens. The scotoma, too, may diminish in size from absorption of extravasated blood, or increase, as the subsequent exudation becomes denser and more copious.

Treatment.—The successful removal of foreign bodies from the vitreous and back of the eye was, until comparatively recently, of so excessively rare occurrence, that practically the chances of an operation undertaken for that purpose being successful were *nil.* At the present time, by the aid of the magnet, first regularly introduced for this purpose by M'Keown, a good many eyes have been saved which must previously have been lost. By this means only particles of iron and steel can, under favourable circumstances, be removed, while, with regard to other bodies, we are not better situated than before. Iron and steel particles form, however, a large proportion of all the cases met with, although the exact proportion differs very much with the locality. The proportion is besides altogether greater than formerly, owing to the great falling off in the number of such accidents caused by the bursting of percussion caps.

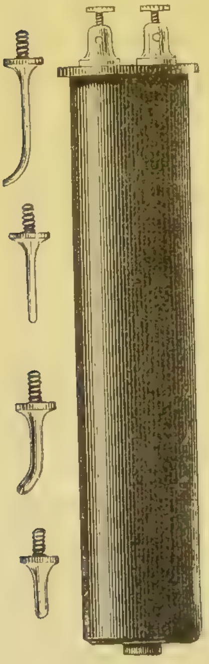

FIG. 94.

The electro-magnet naturally soon became the form in which the magnetic force was applied for operative purposes. Snell's electro-magnet (Fig. 94) is thus described by himself:—"It consists of a core of soft iron, around which core is placed the coil of insulated copper wire, and this again is enclosed in an ebonite case. To one end of the instrument are attached the screws to receive the battery connections, at the other extremity the core of the magnet projects just beyond the ebonite jacket, and is tapped, and into it is screwed a needle, which fits closely on the end of the instrument by a projecting cap." Snell uses differently-shaped probes to screw on to the magnets, and this plan has been adopted in other forms of electro-magnets. Snell's magnet attached to a quart bichromate cell, is capable of lifting from the end of a 2-inch probe a weight of 175 grms. It is of ample power for any use to which it may be put. Hirschberg employs a similar magnet, supplied by five cells, weighing over 1 lb., and capable of raising a weight of 570 grms.

The following excellent rules for the use of the magnet, when the iron or steel lies in the vitreous, are given by Hirschberg. If the wound in the sclera, through which the piece of metal has penetrated, be still open, the magnet should be at once introduced through it. It is generally advisable to enlarge it slightly, so as to admit of the easy removal of the metal adhering to the magnet, and not risk its being rubbed off on withdrawal. Fresh openings may, if necessary, be made in two situations; either by means of a meridional cut through the equatorial part of the sclerotic coat, or by section through the corneo-scleral margin, the vitreous being reached after removal of the lens, or if it be absent, after perforation of the lens capsule. Hirschberg distinguishes three stages after the accident, in which an operation may be undertaken: (1.) The primary stage, that is, before inflammatory symptoms set in; (2.) the secondary stage, in which such symptoms have more or less developed; and (3.) the tertiary stage, when fresh irritative symptoms have appeared after a longer or shorter period of quiescence. Careful probing with the magnet in the first stage, conducted with proper antiseptic precautions, is safe enough, and should be undertaken when the wound is in the sclera, even though there should be some little doubt as to the diagnosis. Otherwise, if there be no evident external wound, and when the diagnosis as to the presence of the metal in the vitreous is not absolutely certain, it is advisable to await the symptoms of the second stage before proceeding to make an opening. If the foreign body can be actually seen in the vitreous, the opening may be made at once, and such cases, of course, afford the best opportunities of obtaining satisfactory results. It is seldom, however, that patients present themselves until the second stage is arrived at, and then the operation should generally at once be resorted to. The section in the sclera should be made at its lower and outer, or lower and inner part. It should be 5-6 mm. in length, and 6-8 mm. from the corneal border. Pieces of iron or steel, up to 30 m. gr. in weight, may be removed during the second stage, and yet leave a chance of a good result. When from 50-180 m. grs. in weight, the only chance is a primary operation, and when much larger, even the successful removal in the primary stage can probably never result in anything further than the retention of the shape of the eye.

In cases of portions of non-magnetisable metals in the vitreous, an operation in the primary stage is hardly justifiable, as the chance, small as it is, of the foreign body being encapsuled, or leading to relatively little irritation, is still greater than that of one's being able successfully to remove it. The removal may be attempted in the secondary stage, however, when the patient has been prepared for the alternative treatment of evisceration or enucleation of the eye, should the attempt fail, as it is likely to do. In any case, the incision should be made radially and not circularly in the sclera, and at the lower part, except where a point of marked tenderness exists in some other situation, when that point should be selected instead. Bodies fixed in the coats of the eye may occasionally be seized with forceps and withdrawn, and a relatively good result be obtained.

Unfortunately the advisability of performing any operation for the removal of foreign bodies from the eye is often questionable, on account of the risk which is then run from sympathetic inflammation occurring sooner or later in the other eye. When the injury has been so severe as to destroy vision from the first, there can be no hesitation about removing an eye in which a foreign body is lodged. If this can be done within the first week, evisceration is the most satisfactory operation—if later on, enucleation. A difficulty in deciding arises in most of the cases where some vision is retained, and here much will depend on the course taken by the inflammation. This is especially the case where the foreign body is a shot pellet. When the penetration has taken place through the sclera and the wound made in it is small, there is not much danger of any septic matter having been introduced at the same time, so that it is generally advisable to make an attempt to keep the eye. Where the exudation can be made out to be extending, removal of the eye should be advised, but an attempt may first be made to extract the foreign body, if the patient thoroughly understands the risk which he still runs, even if this should be successful. Other points in this connection are discussed in the next chapter.

CHAPTER X.

SYMPATHETIC OPHTHALMITIS.

A GREAT number of affections of the eye are, or are supposed to be, in some measure caused by inflammatory conditions which have pre-existed in the other eye, and are consequently looked upon as *sympathetic.* Thus cases of keratitis, choroiditis, neuritis, retinitis, detachment of the retina, and even glaucoma, have been described as sympathetic in their nature. Glaucoma is certainly never sympathetic, and as to the other forms, all that can be said definitely is that their existence is extremely problematical, except in cases where they are at the same time associated with inflammation of the iris and ciliary body. The disease which is generally spoken of as sympathetic ophthalmitis is an inflammation of the uveal tract, most constantly and more especially of its ciliary portion, which comes on—rarely sooner than three weeks, and often much longer—after some injury or affection of the other eye, which has been followed by more or less destructive inflammation in it.

There is nothing absolutely characteristic about a sympathetic ophthalmitis; it may be acute, but it is more often distinctly chronic. It may be mild from the first, or of a decidedly malignant type, and it may be recovered from or lead to considerable or to complete blindness. An inflammation of the eye, which is sympathetic in this sense, is usually very protracted. It is either altogether painless, or accompanied by but little pain. The insidious and dangerous nature of the disease was first pointed out by Mackenzie about the year 1827.

In connection with this subject it is convenient and customary to use the following terms. The eye first affected is called the *exciting* eye, or shortly the *exciter;* the other the *sympathising* eye, or the *sympathiser.*

The inflammation in the sympathising eye, although its progress may be slow, and unaccompanied by well-marked external signs of inflammation, gradually gives rise to all the changes, and is very liable to be followed by the worst consequences of, a severe irido-cyclitis. The first symptom that is often noticed is a defective degree of accommodative power, causing a difficulty of seeing things distinctly near at hand. Some turbidity of the aqueous humour, most noticeable in the area covering the pupil, may frequently be made out about the same time. The iris then loses its glistening appearance and becomes muddy and discoloured, while synechiæ form between it and the lens capsule. There is tenderness on pressure over the ciliary region, the vitreous humour becomes clouded with opacities, and the tension of the eye, which may at first be increased, becomes diminished. A new formation of vessels takes place in the iris, which assumes a greyish colour, and a tumefied, unhealthy, spongy, and, to the practised eye, *rotten* appearance. A membrane soon forms across the pupil, the anterior chamber becomes shallowed, while the periphery of the iris is often to some extent retracted. In some cases there is found to be restriction of the field of vision in a more or less concentric form, due probably to extension of the inflammation from the ciliary body to the choroid. Detachment of the retina gradually takes place owing to the traction of the exuded matter in the vitreous, and leads to complete blindness. But the inflammation may subside before this stage is reached, and a certain amount of vision be retained, along with the appearances of a greyish, thickened, and at the same time atrophic, iris.

Sympathetic ophthalmitis sometimes takes the form of a serous irido-cyclitis from the first. When the inflammation in these cases subsides, as it is more likely to do than in the malignant plastic form, fair or even very good vision may be retained, but even such cases may go on to complete blindness.

Some cases are preceded by what is called *sympathetic irritation*,—that is to say, that while as yet there are no signs of actual inflammation, there may be photophobia, lachrymation, and pain referred to the back of the eye and to the forehead. This irritation is in all respects similar to that experienced in the other eye when a foreign body lodges in the opposite conjunctival sac, or causes some abrasion of the cornea. When to these symptoms of irritation are added circumcorneal injection, there is reason to fear an attack of sympathetic ophthalmitis.

But the symptoms of sympathetic irritation may continue to exist without ever passing on to the dreaded inflammation. On this account it is of importance to make a clear clinical distinction between irritation and sympathetic ophthalmitis. Sympathetic irritation is at once cured by removal of the exciting eye, while such is by no means the case in sympathetic ophthalmitis.

As a general, if not invariable rule, we may take it that where cyclitis of one eye sets up a sympathetic cyclitis in the other, the exciting eye has either never come properly to rest since the accident or disease which caused this inflammation, or else fresh irritation has begun after a period of more or less prolonged quiescence. The renewed inflammation may, it is true, be accompanied by not very marked symptoms, but there will always be found some degree of tenderness to pressure over the ciliary region. Often there is, besides this evidence of cyclitis, circumcorneal injection, lachrymation, and sensitiveness to light. A reinflammation is a common occurrence in cases where foreign bodies are lodged in the eye, owing to their having spontaneously, or through some possibly very slight accident, become dislodged from their encapsuled position. This possibility has therefore to be borne in mind in all cases where a foreign body is known to be retained in the eye, even although comparatively trifling irritation may have followed the accident. As long as the eye remains quiescent there is little or no danger, but the patient must be warned of the risk he runs in keeping the eye, and be urgently cautioned to seek advice as soon as tenderness or redness should at any time manifest themselves. Blows, too, on previously injured or diseased eyes are liable to set up fresh inflammation, and thus lead to sympathy.

The Committee appointed a few years ago by the Ophthalmological Society to arrange a collective investigation on the subject of sympathetic ophthalmitis, came to the conclusion that, in some instances in which a long interval separates the inflammations of the exciting eye and that of the sympathiser, the former might at the time be, and have for long been, perfectly quiescent. The possibility of such being the case should therefore be borne in mind, though more evidence may be reasonably required before it can be accepted as an undoubted fact.

The condition of the exciting eye at the time of its causing sympathy is one not merely of inflammation alone, but of inflammation which is not sufficiently severe to cause purulent destruction of the tissues, and which at the same time is not accompanied by so much serous discharge as to give rise to prolonged increased tension. Sympathetic ophthalmitis very rarely results from panophthalmitis, and all the more rarely the more complete has been the destruction by the purulent inflammation. A glaucomatous condition of the eye, too, whether arising from increased secretion or impeded excretion of fluids, is very rarely associated with a condition liable to excite sympathetic inflammation. *A soft eye, in which perforation, either from accident or ulceration, has at any time taken place, and which is tender to pressure, is that which endangers the safety of the other.* Often such eyes are at the same time blind, but this is not always the case, although it is rare that they have retained any considerable degree of visual acuity.

Sympathetic ophthalmitis is most frequently the result of some injury to the other eye; the wounds which give rise to it may occur in any part of the eye. Wounds in the ciliary region, or extensive cuts involving the ciliary region, and at the same time leading to considerable loss of vitreous humour, are the most dangerous in this respect. Mere corneal wounds may be followed by sympathy, but in such cases they are probably always sufficiently severe, either in extent or from the manner in which they have been inflicted, to give rise to more deep-seated inflammation in the eye.

The excitation may also arise from operations performed on the eye. Of these causes the most common are extractions of cataract, especially in such cases where from encleisis of the iris or vitreous the healing process has been slow and accompanied by considerable inflammatory reaction. Other operations occasionally followed by sympathy are — needling for after-cataract, iridectomy, abscission of staphylomata, &c.

Of the spontaneous diseases of the eye which produce ophthalmitis in the other, the most common are the various forms of perforating ulceration of the cornea, especially when such perforation is followed by more deep-seated inflammation and shrinking of the eye. Curiously enough, however, *a perforation which has arisen from within is not so liable to be followed by sympathetic ophthalmitis as one which has taken place from* *without.* Intraocular tumours, when they set up cyclitis, may also occasion sympathy, though this probably does not occur until they have reached the third stage, and produced perforation of the coats of the eye.

The question as to whether or not true sympathetic inflammation ever occurs, where there has at no time been perforation of the eye, is one which can hardly be said to be yet decided. It is an important question in connection with the pathology of the disease, as will be seen when this is discussed. All cases in which a destructive form of cyclitis or other uveitis occurs first in one eye and then in the other, are of course not necessarily of a sympathetic nature. The real difficulty in all non-traumatic cases is to determine whether the second eye is affected on account of the first—that is, whether the condition of the first eye actually sets up that of the second, or whether the disease in both is not due to the same cause, as so often happens in iritis, in glaucoma, cataract, and other affections of the eye. Only in cases where the first eye has been injured without being perforated, and become inflamed, and been subsequently followed by cyclitis in the second eye, could the presumption be greatly in favour of the sympathetic nature of the cyclitis in the second eye.

From Mooren's statistics I find 125 cases of sympathetic ophthalmitis in which the exciting cause is given. Of these the eye was injured or perforated in some way in 93. In 27 of the 93 an operation had been performed—sixteen times couching, ten times extraction of cataract, and in one case for traumatic cataract. In 20 more out of the 125 cases the exciter was shrunken at the time of the examination, and of these it is only expressly stated with regard to three that there was no trauma. It is not clear from the diagnosis given in each case of phthisis bulbi whether or not this condition was preceded by any perforation. Of the remaining twelve, eleven had some form of complicated or uncomplicated uveitis, and one detachment of the retina alone. Mooren's statistics include a number of other forms of sympathetic affections. These, as explained at the beginning of this chapter, are purposely excluded in our consideration of the subject of sympathetic ophthalmitis, partly because their sympathetic nature is by no means always absolutely clear, but mainly because they do not possess the same importance from a clinical and practical point of view, as does the more common and more serious form. Of his cases of sympathetic irritation, twenty-one are useful for reference, as the exciting cause is given. Eighteen were produced by perforating injuries or inflammations, while in the remaining three, although there was cyclitis, it was not stated whether or not it was set up by injury.

The conclusion arrived at by the Committee of the Ophthalmological Society already referred to was "that sympathetic ophthalmitis occurring without a perforating lesion of the exciting eye is, if not unknown, at any rate extremely rare." They were unable to find any undoubted cases of sympathy without perforation, the nearest approach to this being a few cases of intraocular tumour. Such cases might, however, be deceptive, as it is well known that tumour growth is apt to occur in the site of some old injury.

It is not quite clear whether sympathetic ophthalmitis is more likely to occur in some individuals than in others. According to some writers there is a greater tendency to it in young individuals, whilst others again have held the opposite. Gunn has pretty conclusively shown that there is little foundation for the former belief, and indeed that if there is any difference at all, it is probable, when due allowance is made for the relative number of accidents, that the liability is greater in advanced life. Mackenzie believed—but without, I think, any very good reasons—that it is more frequent in individuals addicted to the excessive use of alcohol and tobacco.

The interval which elapses between the injury to the first eye and the outbreak of sympathetic inflammation of the second is very variable. It is rarely less than three weeks, and may, as has several times been observed, be longer than thirty years. Gunn's statistics show a preponderance of cases occurring between the third and fourth week. On the other hand, the committee on sympathetic ophthalmitis only found eighteen cases out of over two hundred in which the interval was four weeks or less.

This proportion is certainly altogether too small, and is evidently due to a want of attention on the part of the contributors to this point, as in fact comparatively few answers were received to the questions formulated by the Committee regarding the duration. Of these eighteen cases, six began after four weeks, six after three, two between two and three weeks, and four two weeks or less after the accident. Two of these last are certainly doubtful; still it is important to remember that an interval of not more than ten days may very exceptionally intervene between the primary and secondary lesions.

The influence of the length of interval before the outbreak of the inflammation in the sympathising eye on the severity as well as eventual issue of the disease, was another point to which the above-mentioned Committee directed their attention, the conclusion arrived at being that the prognosis is, on the whole, better when the disease comes on after a short than after a long interval. The accompanying table, which is given by Gunn, shows the relation between the length of the interval and the result as well as the character of the inflammation. A short interval was one under a month, a moderate one from a month to a year, and a long one more than a year.

Interval	A.		B.		C.	
	Mild or Good.		Moderate.		Severe or Bad.	
	Inflam.	Result.	Inflam.	Result.	Inflam.	Result.
Short,	,,	,,	3	1	2	4
Moderate,	4	4	15	8	11	18
Long,	1	1	6	3	1	4

From this table Gunn concludes "that the interval appears to have little or no relation to the severity of the disease in these cases; perhaps the tendency to a bad result may be said to have been rather greater with a short than with a moderate or a long one." This conclusion is therefore the reverse of that come to by the Committee. We may take it for granted, then, that if any difference in this respect exists at all, it is practically unimportant.

So far as any practical considerations are concerned, there is no difference in the severity or result of the sympathetic inflammation, whether a short or a long interval intervene between the primary lesion and the characteristic changes in the sympathising eye.

Gunn has found the type of inflammation, as well as the result, relatively severe after operations. His statistics also show that the position of the injury in the case of accidental wounds could not be shown to have any influence on the character of the inflammation or the result. He remarks that "wounds of the ciliary region had as low a percentage of severity as those confined to the cornea, with implication of the iris. Similarly, the nature of the instrument appeared to make no difference in this respect."

Treatment.—In the treatment of sympathetic ophthalmitis we have to consider what can and might in any particular case be done in order to prevent the occurrence of sympathy—that is to say, the prophylaxis—and also what is to be done when the disease has already shown itself, both with respect to the exciting as well as the sympathising eye. There are few diseases in the whole range of surgery in connection with which such difficulties may arise as to the right course of treatment to be adopted. The complete removal of the exciting eye has been hitherto the main prophylactic treatment adopted, although other operations have from time to time been substituted for it, such as evisceration of the globe, section of the optic and ciliary nerves, or of the ciliary nerve alone.

There can be no doubt that the removal of the one eye is capable of preventing the affection of the other eye, which would otherwise take place. If it always did so, much of the difficulty in dealing with cases liable to excite sympathetic inflammation would disappear. But it occasionally happens, though perhaps less frequently relatively than some suppose, that the other eye is affected after complete removal of the injured or diseased one. As, however, it is very easy to overlook the first onset of inflammation in the sympathising eye, it is more than probable that many cases recorded as first beginning after enucleation have in reality begun before, and escaped observation. In two cases of this nature in which I had an opportunity of observing the onset of the sympathetic ophthalmitis, the outbreak in the second eye took place only a week after the removal of the first. In the one the enucleation was performed six weeks after injury; in the other, three weeks after renewed inflammation had begun in an eye injured three years before. I have met with several other instances of sympathetic ophthalmitis appearing first after enucleation, but have not had an opportunity of determining the exact time which elapsed before the inflammation began. In thirty cases discussed by the Committee, already several times referred to, the interval between the enucleation and the appearance of sympathetic disease was under three weeks in twenty-five cases, and more than three, up to as long as eight weeks, in five. The practical point to be gathered from this is, then, that *if three weeks have elapsed since enucleation there is still a possibility, but extremely little probability, of the remaining eye, if it has previously been sound, becoming affected, while after two months it may be considered absolutely safe.*

The sympathetic inflammation never occurs if the first eye be removed within twenty-four hours after it has been injured, and very rarely, if ever, if the operation is performed within the first week. The decision, therefore, so far as the surgeon is concerned, is not so difficult to come to if the case should happen to come under treatment from the first, and if the accident has been so severe as of itself to cause complete and permanent loss of sight. There can, then, rarely be any doubt of the necessity for enucleation, and under these circumstances, too, when the patient is informed of the risk which would afterwards be run by the remaining eye, it is seldom that he will not give his consent. In cases, however, where some, and still more in cases where considerable sight remains after an accident, it may not be so easy for the surgeon to decide on operating, or, if he should deem removal of the eye necessary, to obtain the patient's consent.

The desirability of enucleating in any particular case will depend on the severity and extent of the injury received, as well as on whether or not there has been a likelihood of the introduction of septic matter into the eye at the same time, or shortly afterwards. All wounds involving the ciliary region, and made with some dirty instrument, especially if they have been sufficiently extensive to allow of the escape of vitreous, are so dangerous that an immediate removal of the eye should be urged. Cleanly cut wounds with anything which is itself clean, such as steel or glass, are less dangerous, and when the vision is not destroyed, an attempt should be made to keep the eye, by avoiding as much as possible everything which would be likely to set up, or keep up, inflammation. Thus the wound should be treated antiseptically in the manner frequently described already, the eye should be bandaged, and the other shaded, all reading and strong light avoided, &c. When foreign bodies have penetrated the eye, an attempt may be made to remove them if they have not led to destruction of vision, or are not likely to have carried septic matter with them. If this attempt fails—and it is most likely to do so, except in the case of portions of steel and iron—or if the symptoms of irritation to which their presence gives rise do not quickly subside, it will be necessary to have recourse to enucleation.

There can be little doubt that the fear of sympathetic ophthalmitis has, in this country at least, led to the sacrifice of many eyes, which might have been retained, or have even recovered a useful amount of sight, and never caused inflammation in the other; yet it would take many mistakes of this kind, in which, too, there will always be a doubt as to whether the eye might after all be safely left, to justify the same self-reproach which the opposite mistake must cause in any one who is properly alive to his responsibilities. In many instances a case first presents itself for treatment after some time has elapsed since the occurrence of the accident, or after perforation has taken place from some other cause. In such cases, if the eye is blind, soft, and tender to touch, there can be no question that the proper treatment is to enucleate as soon as possible, as the patient will then have the best chance of escaping a sympathetic inflammation, and is, with the exception of the appearance, as well off as he was before.

The difficulty arises when such an eye has retained some sight. If it is removed, it may just happen that this is done too late, and the other eye become subsequently affected. It should always be remembered that the exciting eye may eventually be the only one in which any sight remains; it may even completely recover, and yet the sympathising eye be lost. In nine such cases collected by the Committee on sympathetic ophthalmitis the sympathiser was lost five times. When this happens, I believe the proper treatment is to enucleate the second eye, as cases have been met with where there seems to have been good reason to think that it has reacted on the first or original exciter.

If, when the case is first seen at a time when sympathetic ophthalmitis has already begun, the exciting eye is found to be blind, it should be removed, provided the sympathising eye has not long been inflamed—not more than two or three weeks. There seems every reason to believe that, although in most cases it may not do so, enucleation of the exciting eye has a tendency to modify the severity of the inflammation in the sympathiser. This risk must not, however, be run in the case of exciters which have retained any useful amount of vision, for reasons similar to those just explained.

The *treatment of the sympathising eye* is one on which there are many differences of opinion. Mercury is most commonly used, and beyond this, merely the ordinary precautions indicated in all cases of uveal inflammation.

Although the Committee appointed by the Ophthalmological Society could not point to any greater success, on the whole, amongst cases treated with mercury than amongst those treated without, their statistics on this point certainly showed that mercurial treatment did no harm. It is always difficult, of course, to prove the curative action of any drug, yet I should certainly not hesitate to recommend the systematic use of mercury in sympathetic ophthalmitis. There is such a general impression that by pushing mercury to salivation the mercurial treatment has been properly tried, that statistics gathered from so many different sources are in this respect of little value. The fact is, as I have pointed out in the chapter on iritis, that symptoms of mercurialisation must be avoided as long as possible before the effect of mercury can be said to be attained. Cases treated during many weeks by inunction, as can be done with proper care, are, I believe, as a rule benefited by it.

Atropine should be used from the first, but the same risks apply as for iritis from other causes; when synechiæ have formed, and the use of a mydriatic appears, from the dragging which it causes on the attachments, to give rise to irritation, it has to be discontinued.

It must be admitted that the question as to whether or not an iridectomy should be performed is one of considerable practical difficulty. Some authorities do not recommend it under any circumstances; others again perform the operation in all cases where the eye has come to rest, but warn against it as long as there is any irritation. There is a great tendency for the coloboma formed to close up again, and it is undoubtedly the case almost always that any operative interference only makes matters worse. One thing there can be little doubt about, if an iridectomy is attempted it should be made as large as possible. The only cases in which it appears to be indicated are cases where the iris is decidedly bombé, and in which, therefore, there is a distension and not an obliteration of the posterior aqueous chamber. Yet even in such cases one has often to regret having interfered at all.

In all cases which are left to settle down, and in which all vision is not lost, although from the extensive deposits of plastic exudation in the pupil it is very defective, the best and safest chance of getting an improvement is to extract the lens, and afterwards, as will always be found necessary, follow this up with an iridotomy, which should only be performed after the lapse of many months.

Some cases of sympathetic inflammation may remain for a long time quiescent, and then recommence, just as is the case with, and probably for much the same reasons as, the similar inflammation in the exciting eye. This must be borne in mind in connection with any operation performed on a sympathetically inflamed eye, even when it has been for months thoroughly quiet, and for this reason it is advisable, when there is any degree of useful vision, not to interfere in any way.

Reference has already been made to other operations on the exciting eye, which have recently been substituted for enucleation. As the efficiency of the operations cannot yet be said to rest on any established practical basis, it must be judged of from a consideration of the most likely pathology of sympathetic disease. From this point of view I should altogether reject section of the ciliary nerves, and regard even the division of both optic and ciliary nerves with suspicion. Indeed, a case of sympathetic ophthalmitis has already been met with by Voelcker, after optico-ciliary neurotomy. Evisceration, however, when performed in time—that is, not later than the first week—would seem to fulfil the indications required in all cases of injury to the eye at all events. At a later period enucleation gives possibly a better chance. The number of times that evisceration has been performed must be very small indeed, compared to that in which the eye has been removed altogether, and yet two cases have already been recorded by Cross, in which changes described as of a possibly sympathetic nature were met with after the operation. This should be remembered, although the fact by no means proves that similar changes might not have followed enucleation.

Pathology.—An explanation of the pathology of sympathetic ophthalmitis, to be satisfactory, must account for certain facts connected with this disease, of which the following are the chief:—(1) Why perforating lesions, and such as cause cyclitis, should be, if not the only exciting causes, at least by far the most frequent; (2.) Why a certain time—rarely less than three weeks—should elapse before the second eye becomes affected; (3.) Why removal of the exciting eye is not always sufficient to prevent its occurrence; (4.) Why some cases only come on long after the first eye has become quiescent; (5.) Why it should not occur with cases of panophthalmitis; and (6.) Why the inflammation so frequently settles in the ciliary body and iris.

One view held by Mackenzie as to the cause of sympathetic inflammation was that the state of irritation of the retina which he supposed to be affected was transmitted to the retina of the other eye by passing up the one optic nerve and down the other. An irritation of the previously sound eye produced in this way might, he believed, be the beginning of the more serious and destructive inflammation. Mackenzie, whilst mainly apparently inclining to this explanation, did, however, admit the possibility of two other channels of transmission, viz., the vessels and the ciliary nerves.

It was Heinrich Müller who first succeeded in directing general attention to the ciliary nerves in this connection; and more or less fanciful explanations of the transmission along this channel seem to have been those which until recently were most generally favoured. Possibly some kind of excitation of the ciliary nerves may account for sympathetic irritation, but it is pretty certain now that the characteristic ophthalmitis is in no way connected with it. On this point I cannot do better than quote the conclusions arrived at by Brailey. Speaking of inflammation of the ciliary nerves, he says: "It is doubtful if it can ever be a cause, since it is not infrequently entirely absent in the exciting and sympathetically inflamed eye, and since, moreover, it is markedly present in some cases of irido-choroiditis, which neither have produced, nor, judging from their microscopical characters, are likely to produce, sympathetic disease. Inflammation *round* the ciliary nerves, though more often present in both the exciting and sympathetic eye, is not an essential cause, since it is usually less marked than the inflammation round the accompanying artery, from which it has evidently spread." It has also been supposed that the coats of the ciliary arteries might transmit an inflammation from the one eye to the other. This would certainly be a more direct course for the inflammation to take. Brailey's investigations have also shown this view to be untenable, as the inflammation may be absent in cases which give rise to sympathetic inflammation, and is not very uncommonly met with in panophthalmitis, after which sympathetic disease is almost unknown.

In 1880 Berlin suggested a metastatic explanation. According to his hypothesis, germs which are introduced into the eye owing to its perforation produce by their development and multiplication the characteristic plastic uveitis. They are then taken up by the circulation, and although deposited in different parts of the body, only find a suitable nidus for their destructive development in the uveal tract of the other eye. The experiments adduced in support of this view are not very convincing, and do not clearly demonstrate anything further than the much greater tendency towards the production of severe inflammation in the first eye when septic matter is introduced into it. Five years later a hypothesis, essentially similar to Berlin's, in so far as the transference was supposed to take place through the circulation, was advanced by Hutchinson. He, however, likens sympathetic ophthalmitis "to the process which occurs in the multiplication of malignant new growths through the blood elements, rather than to that by bacillar septicæmia." Apart from the improbability of the products of inflammation being removed by the blood and deposited, and thereby setting up a similar condition of inflammation in a definite part of the other eye alone, this view is not consistent with clinical experience.

In 1879 Knies was led, by the microscopic examination of the eyes and optic nerves in a case of serous iritis, to put forward the hypothesis that sympathetic inflammation is the consequence of the direct transference of products of inflammation along the lymph channels of the optic nerves and their sheaths. He subsequently adduced many reasons, based on clinical as well as pathological investigations, in support of this view, which, although generally disbelieved in at the time, must now be looked upon as having marked the commencement of a new era in the history of the subject. Not long afterwards Knies' hypothesis received the support of such authorities as Horner, Leber, and Snellen, the last of whom, at the London Congress in 1881, expressed as his opinion not only that the transference from one eye to the other took place along the sheaths of the optic nerves, but that in all probability the cause of the symptoms characterising sympathetic inflammation was a transference of germs along this channel. In 1881 attempts were made by Deutschmann to produce sympathetic inflammation in animals. The result of these experiments was to show that germs introduced into one eye of an animal did find their way to the other along the optic nerves, and there set up inflammation; but it was not found possible in this way to restrict the inflammation in the second eye to the uveal tract. Germs were also constantly found in human eyes removed on account of having excited sympathetic inflammation, and even portions of iris excised from sympathising eyes have been found to contain similar germs.

Deutschmann's researches show that some substance capable of exciting inflammation really passes by the optic nerve to the other eye. This substance is probably in most cases a collection of micro-organisms, but it may possibly sometimes be some material resulting from their action on the tissues of the first eye. It thus gives a satisfactory account of the first point mentioned at page 350 as requiring explanation. The second difficulty, again, is readily explained by the passage along this channel necessarily occupying some time, while it is evident that, when the germs have got the length of the nerve, removal of the eye could not be sufficient to arrest the process, which may nevertheless be less severe, as there must be a necessarily less considerable number reaching the second eye when the source of their production has been cut off. In this manner, therefore, a satisfactory explanation seems to be given for the third difficulty. To explain the fourth point—viz., why cases of sympathetic inflammation should often occur long after the original lesion which has given rise to the first inflammation, and therefore long after the presumable introduction of micro-organisms into the eye—it is necessary to assume that it is possible for these organisms to become encapsuled, or in some way prevented from being carried into the lymph channels, an assumption which is not inconsistent with their known behaviour elsewhere. The reason why injuries to the ciliary region are more liable to cause sympathetic mischief, may not unlikely be because that part of the eye, owing to anatomical peculiarities, as well as its being difficult to keep at rest, is more subject to prolonged inflammation. There are, besides, in connection with it lymph channels which communicate with those of the optic nerves. When an inflammation, even though produced by micro-organisms, is excessively severe, and results in purulent destruction of the tissues, as in the case of panophthalmitis, the lymph channels become obliterated, and the danger of further transference of the organisms averted.

The immunity given then by panophthalmitis—a clinical fact which has long been observed—is explained without too great stretching of this hypothesis. The last difficulty, it must be confessed, has still to be cleared up, although it is easy to frame a hypothesis consistent with the theory of germ-transmission along the optic nerve, which appears a satisfactory explanation. Thus it is a well-known fact that these minute organisms give rise to the most evident signs of inflammation at the point where they tend to accumulate in the greatest numbers, and it does not seem improbable that the migrating organisms may pass on without, as a rule, causing marked disturbances, until they become arrested at the ciliary body. This, however, requires proof, and until this proof is furnished, the whole question cannot be said to be definitely decided.

Others, and notably Gifford, who have repeated Deutschmann's experiments, have had occasion to modify the theory with reference to the exact channels along the nerves which are taken by the migrating germs, but the main results have been pretty well confirmed.

This recent elucidation of the pathology of sympathetic disease cannot but exercise some influence on the treatment of such cases. Thus it becomes evident that the operation which enables one to remove the largest portion of the course along which the infecting matter travels must afford the greatest chances of success. Hence the advisability of performing enucleation instead of evisceration, except at the very beginning, before it is likely that migration has begun. Further, it points to the great importance of careful antiseptic treatment of all perforating wounds and ulcerations. There can be little doubt, too, that prolonged mercurial treatment, taking into consideration the action of mercury as a germicide, is indicated, while every means of allaying irritation, and keeping the eye, especially the ciliary muscle and iris, at rest, cannot but tend to exercise a favourable influence on the exciting process.

CHAPTER XI.

GLAUCOMA.

The disease now recognised as glaucoma is, on account of its relative frequency, its disastrous course when left alone, and above all its curability if treated at an early stage, perhaps the most important in the whole range of eye diseases.

The chief objective signs of glaucoma are—(1.) Increased tension; (2.) Dilatation and immobility of the pupil: (3.) Haziness of the cornea: (4.) Shallowness of the anterior chamber: (5.) Enlargement of episcleral veins: (6.) Excavation of papilla: (7.) Visible pulsation of the retinal artery.

At the same time the following subjective symptoms are met with;—(1.) Defective visual acuity: (2.) More or less characteristic limitation of the field of vision: (3.) Photopsia: (4.) Anæsthesia of the cornea: (5.) Pain.

A marked and important characteristic of glaucoma is the intermittent nature of both objective and subjective symptoms. This intermittency is met with at all stages of the disease, and even persists when the eye is totally blind, subjective light sensations and pain coming on every now and then, and at other times being wholly absent.

Glaucoma is met with in different forms, and various classifications of such forms are adopted by different writers. From a clinical point of view the following forms are usually distinguished:—

A. Primary glaucoma.
  1. Inflammatory glaucoma.
    (*α*) Acute. (*β*) Chronic.
  2. Simple glaucoma.
B. Secondary glaucoma.

Inasmuch, however, as there cannot be said to be any real inflammation characteristic of glaucoma, it seems better to use the term *congestive*, as is done by Priestley Smith, instead of inflammatory.

Clinically, we may distinguish three stages of the disease, in whatever form it is met with :—(1.) the *threatened* stage, in which the premonitory symptoms to be shortly described assert themselves; (2.) the *confirmed* stage, in which the disease has led to defective vision, which gradually increases until (3.) the glaucoma is said to be *absolute*, or has resulted in complete destruction of vision. In many cases degenerative changes have already begun by the time the absolute stage is reached; in all cases such changes eventually make their appearance.

Primary glaucoma is essentially a disease of advanced life; it is rarely met with before the age of forty, and increases in frequency with age. It is more common in some countries than in others. In this respect there is a curious difference between England and Scotland, the disease being considerably more frequent in England. Among 10,000 consecutive cases treated in the Eye Department of the Edinburgh Royal Infirmary, 86 were cases of glaucoma—45 in women and 41 in men. It is more common amongst Jews than Christians.

It is most common for both eyes to be subject to attacks of glaucoma. This is in no way a sympathy, but depends upon the fact that the conditions which are apt to lead to the disease usually exist in both eyes.

The most common state of refraction met with along with glaucoma is hypermetropia. It has been pointed out that a greater proportion of eyes with glaucoma are hypermetropic than can be accounted for by the proportion of hypermetropic compared with other states of refraction altogether. This has by some been explained by supposing that the disease tended to render the eye hypermetropic; by others, on the other hand, by admitting that hypermetropic eyes, owing to rigidity of the sclera, are more disposed to glaucoma. The first view has been pretty well proved by actual measurement and experiment to be incorrect. As to the second, it is not easy to say in how far it is justified, but it must be remembered that in advanced life, when glaucoma usually occurs, acquired hypermetropia is very common.

In a certain proportion of cases of glaucoma, variously estimated at from one-third to three-fourths of all cases, the actual outbreak of the disease is preceded by premonitory symptoms, more or less marked, and occurring at, as well as extending over, longer or shorter periods of time. What distinguishes the disease in this stage from that in which it may be said to be fully developed is, that during the intervals between the occurrence of these symptoms the functions of the eye are to all appearance normal. The only manner in which it may be found abnormal by the ordinary methods of examination is in the frequent existence of a higher degree of presbyopia than that which corresponds to the age of the individual—a condition which should consequently, and even when no premonitory symptoms have been observed, excite suspicion.

The *premonitory symptoms* are obscurations of vision, and the appearance of haloes round lights. The vision appears more or less misty and veiled, this being especially noticeable for objects which are not very strongly illuminated. At the same time, if the flame of a candle or gas jet be looked at against a dark background, it is seen to be surrounded by a colourless space, which is encircled by a coloured halo, the intensity of which differs in different cases, as well as at different times in the same case. These symptoms usually come on after some mental or physical fatigue, *e.g.*, a sleepless night, or prolonged fasting. They disappear after a meal, and more constantly after sleep, and can also almost invariably be cut short by the use of a myotic. The intervals between such attacks are very variable, often amounting to months or years, but usually having a tendency to become shorter and shorter, until the glaucoma suddenly or gradually passes from the premonitory to the confirmed stage.

Premonitory symptoms appear to be on the whole more common the younger the individual attacked with glaucoma. The cloudy vision varies in amount, but may be very disagreeable to the patient at a time when he is found on examination to have full visual acuity, although as a rule it is accompanied by some diminution in the acuteness of vision. The halo seen is always perfectly circular. Its apparent size increases, and the breadth of each coloured ring becomes greater the further the light is from the eye.

According to Laqueur, who made a number of perimetric measurements of it, the diameter of the halo is pretty constantly from 10° to 11°, while Donders' measurements only give 7°. Measurements which I have made confirm those of Donders. From 7°-8° may be taken as the usual diameter. The intensity of each colour, apart from individual differences in the luminosity of the flame round which they are seen, depends on the character of the light. Round candle and gas flames the red hues are the strongest, whilst the bluish ones predominate round the electric light. The halo is independent of the direction from which light falls into the eye, and is therefore seen as well, though not with the same distinctness, round objects situated eccentrically with respect to the line of vision. In some cases radiating striæ are seen besides the coloured rings. The colourless space which immediately surrounds the flame is light near the flame, and becomes darker and darker the nearer it is to the rings of colour. These again are always arranged in the same order, the most external ring being red, the most internal bordering on the black space, bluish or bluish-green.

During the premonitory attack the tension of the eye is always increased and there is more or less diffuse opacity of the cornea. Sometimes, however, the opacity is so slight as to be barely noticeable on oblique illumination. There can be little doubt that the haloes are due to the diffraction of the rays passing into the eyes, and this diffraction probably mainly takes place in the cornea as the result of the opacity, the nature of which is described later on.

This was proved by Donders as long ago as 1850. He found that, on covering the lower half of the pupil, the outer and upper, and lower and inner quadrants of the halo disappear, while on covering the upper half the other quadrants disappear. The prismatic arrangement of the colours, the fact that they are uninfluenced either by accommodation or the movements of the eye, as well as the occasional appearance of a second halo with the same distribution of colour as the first, are further confirmations of the truth of Donders' explanation.

The obscurations of vision are also to a great extent, though probably not entirely, due to the opacity of the cornea. To some extent, varying most likely in different cases, they appear to depend on the alteration which takes place in the choroidal and retinal circulation, by which the light sense is diminished. This explanation is suggested by the more marked veiling which is experienced by the patient in subdued light, as well as by the not altogether infrequent occurrence of misty vision when the cornea is to all appearance clear.

The vision of haloes round lights is not altogether pathognomonic of glaucoma. It is met with, for instance, in cases of conjunctivitis, owing to the diffraction caused by the conjunctival secretion lying in front of the cornea. In such cases the haloes disappear on rubbing the eye. A good many people, too, with normal eyes see them, especially if the pupil be dilated. They are probably, however, rarely seen of the same intensity as in glaucoma, so that when suddenly observed they should awaken suspicion.

If an ophthalmoscopic examination be made at the time when the premonitory symptoms are complained of, there is sometimes seen to be a spontaneous pulsation of the arteries on the disc. This appearance disappears with the disappearance of the subjective symptoms, and of the increase of intraocular tension. The same pulsation may be produced by external pressure on the eye, and in cases where the intraocular tension is increased, and yet the pulsation not visible, it may often readily be elicited by slight pressure on the eye with the finger.

One cause of the pulsation, therefore, appears to be the interference with the arterial blood-current resulting from the abnormal degree of tension within the eye; but it is probable that in most cases where it occurs spontaneously there is, besides, some disease of the arteries, which may or may not be associated with diminished blood pressure.

The *increased intraocular tension* is not always present at the time of examination of an eye affected with glaucoma. During an attack when the cornea is hazy, the vision more interfered with than usual, and the patient suffering pain in the eye and over other parts in the region of distribution of the fifth nerve, increased tension is rarely if ever absent. In the non-congestive form of glaucoma it is usually less constant and less pronounced than in that in which there exists an evident congestion. The degree of intraocular tension is subject to considerable differences physiologically, some eyes being distinctly harder than others, and for this reason it is not by any means easy to feel sure in any particular case that the tension is higher than normal. If, for other reasons, one is led to suspect a possible increased tension in one eye only, it should be compared carefully with the other, which affords the best standard for judging of the tension in the doubtful one.

Various instruments called tonometers have been devised for the purpose of measuring the intraocular tension, but none of these appear to afford any more delicate or more trustworthy information that is given by palpation with the ends of the fingers. Two ways of making such palpation may be employed. The point of each forefinger may be placed on the closed eye, and alternately pressed gently upon the globe through the upper lid; or the forefinger of one hand may be applied directly to the conjunctiva covering the sclera when the eye is directed upwards, and a series of slight taps given, without at any time removing the finger altogether from contact with the eye. In either of these ways the resistance of the eye can be determined with considerable delicacy, so that a little practice suffices to enable one to gauge the relative hardness of two eyes tested at the same time. It is more difficult to acquire the power of estimating the amount of change in tension. It is customary to distinguish three degrees of increased hardness. These three degrees, which were pointed out by Bowman as practically distinguishable, are—(1.) A marked increase, as compared with the normal, or T + 1 according to Bowman's notation; (2.) A great increase, but one in which, to the palpating finger-points, the eyeball still admits of some dimpling of the globe, T + 2; and (3.) That degree of tension upon which no, or little, impression is made even by firm pressure on the eye, T + 3. A doubtful increase may be noted as T + ?

While such a division is extremely practical and universally adopted, it must not be supposed that any hard and fast line exists between the different degrees of increased tension; thus it is not an uncommon thing to find a degree of tension which would be denoted by one examiner as T + 1, and by another equally practised as T + 2. This is, however, of course a matter of little importance, as under such circumstances there is no doubt as to the abnormal degree of tension. The more important cases are those in which there may be a difference of opinion as to whether any increase in tension does or does not exist.

Dilatation of the pupil, along with more or less complete immobility, is very constant in glaucoma. The dilatation is not always regular, and the pupil is frequently oval or egg-shaped, owing to greater dilatation at some parts than at others. This is a point of some importance in connection with the differential diagnosis between simple, non-congestive glaucoma and optic atrophy. The eye presents at the same time a muddy appearance, and the pupil frequently has that greyish-green colour which gave rise to the name of glaucoma. This is due to the reflection of light from the surface of the lens, modified to some extent by the opacity of the cornea, and probably also the aqueous humour, and apparent on account of the dilatation of the pupil. The pupil may be got to contract by the use of eserine, and to dilate still further by atropine, in most cases where the iris tissue has not undergone an atrophic change—most readily therefore, on the whole, in early cases. There is consequently no real paralysis, so that the term iridoplegia which is sometimes used to express the condition of the pupil, is hardly applicable.

It is generally assumed that the dilatation is the result of a paresis of the ciliary nerves to the iris caused by the increased fluid pressure within the eye. Schnabel has, however, pointed out very correctly that this view necessitates the unwarrantable assumption that one set of fibres within the eye is paralysed by pressure, whilst others escape. There are certainly no anatomical reasons for supposing that the dilator nerve fibres should escape a pressure which would tell on those supplying the sphincter pupillæ, nor has it been shown that, given an equal degree of compression of both sets of fibres, the function of the latter should sooner and more completely be interfered with than that of the former. There is further the difficulty, which von Graefe recognised, of explaining on this assumption the frequent irregularities met with in the dilated pupil. A dilated pupil is found, too, in cases where the tension is not distinctly increased. Besides, the dilatation often unmistakeably precedes other symptoms of glaucoma. When the pupil is dilated there is a corresponding constriction of the vessels of the iris. It is not improbable, therefore, that the dilatation which takes place in glaucoma may be due to the occurrence from some cause or other of such a vascular constriction. On the other hand, Priestley Smith has shown that one can by pressure on the eye produce dilatation of the pupil.

The haziness of the cornea is a constant appearance during the period of exacerbation in all congestive cases. In chronic non-congestive cases it is often absent, or present only to a very slight extent, though at times, when the tension is high, it may even in such cases be sufficiently evident. The peculiarity of the corneal opacity in glaucoma is that it is uniform and most intense toward the centre of the cornea. It is always accompanied by a dull and somewhat stippled appearance of the surface. A characteristic, too, is the disappearance of the opacity immediately on or very soon after the return of the tension to the normal.

The cause of the opacity was long ago rightly referred to œdema of the cornea by Arlt, but it remained for Fuchs to give the anatomical demonstration of the œdema. This he did in the case of eyes enucleated for sarcoma in which there were glaucomatous symptoms. Vertical sections through the cornea in such cases show spaces between the lamellæ, which are either empty or filled with a coagulated fluid. These spaces are more numerous towards the anterior part of the cornea. In such sections coloured with carmine numerous fine dark lines may be seen traversing Bowman's membrane from behind forwards. These are not seen in non-œdematous corneæ, and correspond to the dark lines which are to be found in sections stained with chloride of gold. They are in fact the nerve filaments which pass outwards to the corneal epithelium. They are visible in the œdematous cornea on account of the deeper staining of the coagulated fluid which passes along the nerve channels to reach the epithelial cells between and behind which it is found to lie. It is this position of the fluid which gives the dull stippled appearance to the surface of the cornea. Fuchs' preparations leave no doubt that the fluid passes by the nerve channels. He considers, too, that it coagulates as soon as it comes into contact with the epithelial cells. This cannot be the case, as a general rule at all events, as it is well known that the haziness generally immediately clears off after the performance of iridectomy. There is probably always some fluid between the corneal lamellæ, but it is only when the intraocular pressure is increased that a stasis in and a distension of the spaces occur, with a still further pressure into the epithelial layer, and a disappearance of the œdema on the return of the normal tension is due, no doubt, to the elasticity of corneal tissue. In old-standing cases a permanent form of opacity is met with, which is probably due to some other cause than mere œdema.

Shallowness of the anterior chamber is almost invariably met with to a greater or less extent, though sometimes the opposite condition occurs. The shallowing is due to the pushing forwards of the lens and peripheral portion of the iris; in long-standing cases, also no doubt to the adhesion which takes place between the periphery of the iris and the posterior surface of the cornea, which leads to the blocking up of the angle of the anterior chamber. At times when the tension is great the anterior chamber is often reduced so much in depth that the performance of iridectomy is rendered difficult. In most cases where glaucoma occurs in young individuals the anterior chamber instead of being shallow is abnormally deep. This is also the case in some forms of secondary glaucoma.

Enlargement of episcleral veins occurs in most cases which have existed for some time, more especially in the congestive form of the disease, though this change is rarely absent altogether, even in cases of simple glaucoma. In acute congestive glaucoma we meet with hyperæmia and chemosis instead, as the result of the sudden disturbance in the venous circulation within the eyes, which, when long continued, is the cause of the distension of the external veins.

Excavation of the papilla—the so-called glaucoma cupping —is met with sooner or later in all cases of glaucoma, but the extent of the cupping does not stand in any very close relation to the degree of visual defect. When fully developed, the papilla in glaucoma presents the following appearances on ophthalmoscopic examination. The vessels are pushed to the inner or nasal side, and appear to bend sharply round the margin of the disc in this situation, without therefore coursing over any portion of normal papilla, the veins being at the same time frequently distended and the arteries small. The rest of the papilla presents a greyish-blue appearance, which is seldom uniform, being more frequently specked with white, especially at the centre. There is besides, usually, and when the retinal vessels are not very greatly atrophied, a blurred appearance of vessels to be seen in this bluish area. A few cases of hæmorrhage into the cup have been recorded. According to Loring the appearance of hæmorrhage may possibly be due in some such cases to the formation of numerous new vessels. Surrounding the papilla there

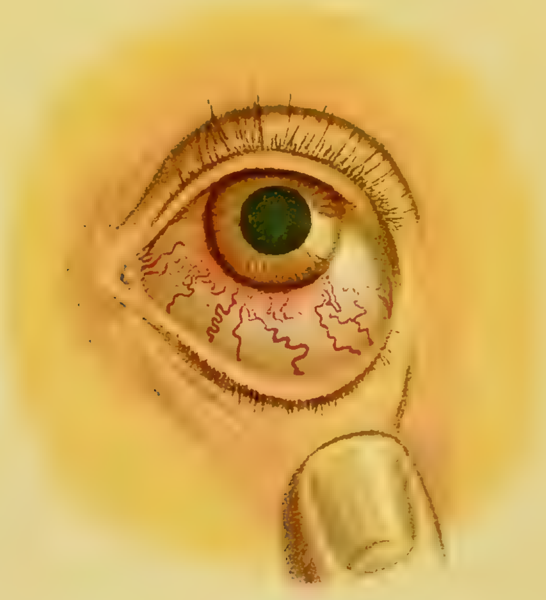

FIG. 95.—Glaucoma, external appearances.

is almost invariably a whitish-yellow ring visible, the breadth of which varies considerably in different cases, but which is seldom very broad—seldom measuring, for instance, one-fourth or one-third the breadth of the papilla—although it is occasionally seen even quite as broad as the papilla. The breadth is as a rule uniform throughout. With ordinary care it is easy to satisfy one's-self that this ring is not a portion of the papilla itself, but an abnormal appearance of the immediate surroundings of the papilla.

These appearances, which were observed soon after the invention of the ophthalmoscope, and the discovery of which led, as has been already said, to a considerable modification in the views as to the nature of glaucoma, were at first taken as representing a swelling or prominence of the papilla instead of an excavation, a mistake which was not long in being corrected by Weber.

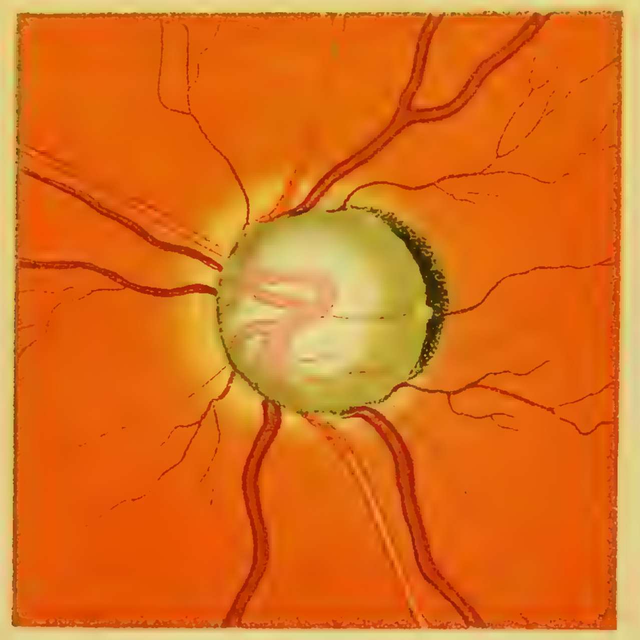

Fig. 96.—Cupping of disc, from a case of glaucoma.

Even a slight degree of excavation can be readily diagnosed by the indirect method of ophthalmoscopic examination, by observing the parallax caused by moving the lens from side to side. The edges of the papilla are in this way seen to apparently slide back and forwards over the central part. From this we know that the aërial image of the borders of the disc lies nearer the observer than that of the centre of the disc, and consequently is that of an object whose distance behind the cornea is less. The actual depth of the excavation can be approximately determined by estimating the refraction by direct ophthalmoscopic examination of the margin and of the centre or deepest part of the cup, and allowing three dioptres to every millimetre. With Giraud Teulon's binocular ophthalmoscope the proper relief is obtained and the excavation evident.

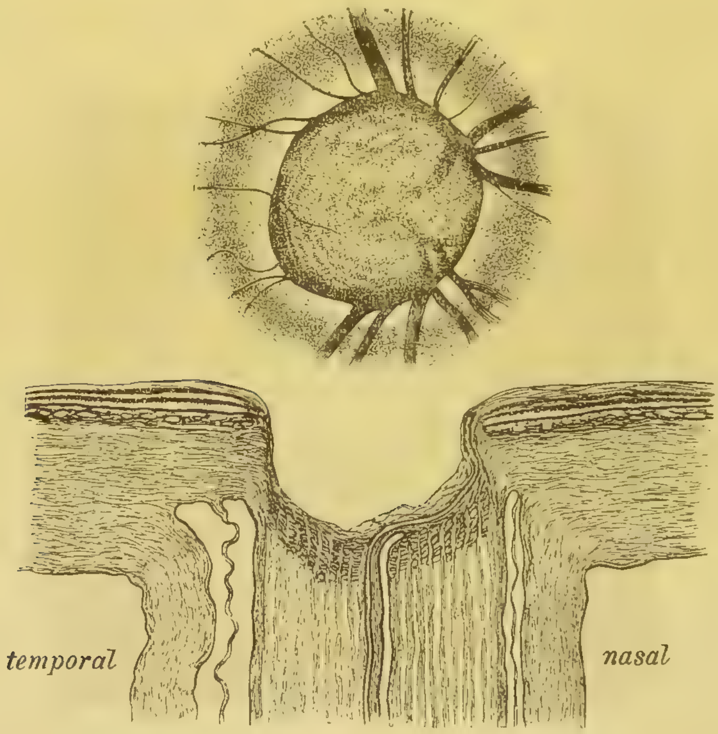

Fig. 97.—Longitudinal section through papilla with glaucomatous excavation, showing cause of ophthalmoscopic appearances (after Fuchs).

There is little difficulty, as a rule, in diagnosing a glaucoma excavation when fully formed, but the less marked changes which occur at first are by no means always entirely characteristic, and may be confounded with physiological excavation, and also with that excavation which is met with in atrophy of the nerve. When the visual defect is inappreciable, the glaucoma excavation may be taken for a physiological one, while, on the other hand, in cases where, from some cause or other, vision is at the same time impaired, one is apt to ascribe undue importance to an existing physiological excavation. The physiological excavation is white, and never involves very much of the nasal part of the disc, over which the vessels can consequently always be seen to course. When large, it may extend over a large part, or even the whole, of the temporal half. The vessels can generally be followed down the side of the excavation, which is funnel-shaped, and not steep, as that met with in glaucoma. In cases of uncertainty we have to fall back upon the subjective symptoms, in order to avoid mistakes. The greatest difficulty is experienced in simple glaucoma sometimes, when the excavation is not very characteristic, as the subjective symptoms are hardly distinguishable from those accompanying atrophy. The atrophic excavation is shallow, and as a rule occupies the whole disc. The vessels never bend sharply over the margin of the disc, and can be seen, more or less out of focus, continually from the point or points at which they emerge from the nerve. The appearances, which of themselves would tend to render glaucoma the probable cause of the excavation, should it be slight and not very typical, are the crowding of the vessels to the nasal side of the disc, and the yellowish ring surrounding the disc. The straight dipping of all the vessels, which is met with in a later stage, is quite characteristic.

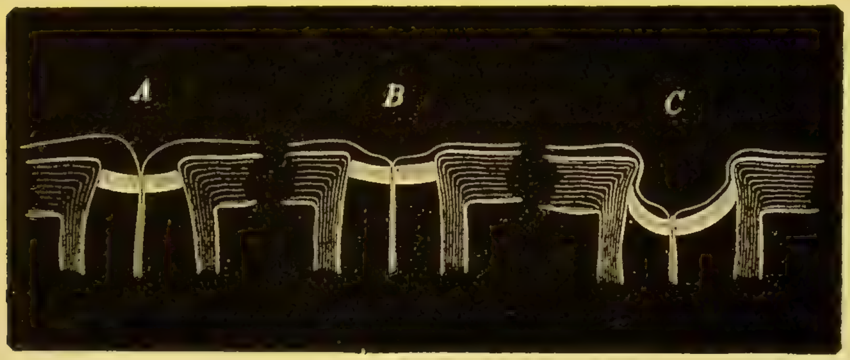

FIG. 98.—Diagrammatic representation of the different forms of excavation. (*a*) Physiological; (*b*) Atrophic; (*c*) Glaucomatous (after Fuchs).

A spontaneous pulsation of the retinal arteries on the cupped disc is almost a certain proof of the existence of glaucoma. Such visible arterial pulsation is very rare under other circumstances. It is due to some resistance to the flow of blood through the retinal arteries, so that the current is only complete during a systole. The impediment is mainly no doubt the result of increased intraocular pressure on the vessels, but it is possible that, in many cases at least, it is due in some measure to an active spasmodic constriction of these vessels themselves. It disappears almost invariably after iridectomy, and cases where this pulsation is visible are such as are most likely to be benefited by operation.

The cause of the excavation of the papilla in glaucoma appears to be the gradual recession of the lamina cribrosa. This structure seems unable to resist for long the increased pressure to which it is subjected, so that after frequent attacks of abnormal tension the end of the nerve becomes hollowed out. The excavation of the papilla depends in all probability less on the degree than on the duration of the increased tension. The yellow ring is generally believed to be due to some atrophy of the choroid caused by pressure or distension as the lamina cribrosa recedes. Some have maintained that the appearance is produced by an inflammatory exudation which takes place in this situation. The great regularity in breadth seems to argue strongly in favour of the distension being the first cause, although it is not unlikely that in some cases inflammatory as well as purely atrophic changes may take place.

It is believed by some that the want of resistance in the lamina cribrosa is the result of inflammatory change, which may be so pronounced as to admit of its recession and the consequent cupping, even where the tension of the eye has at no time risen above the normal standard. If this be the case at all, it is certainly not a common occurrence, most, if not all, excavations being met with where at all events occasional increase in the intraocular tension takes place. It seems not unlikely, too, that there may be an individual difference in the resistance of the lamina cribrosa. Whether the pathological changes which are undoubtedly met with in the nerve in many cases tend to favour this yielding to the increased tension or not, does not appear to be as yet satisfactorily established.

The acuteness of vision begins to diminish and become permanently impaired as soon as the glaucoma has passed from the premonitory to the fully developed stage. During the existence of premonitory symptoms, more or less diminution of vision takes place, but this is recovered from as soon as the attack passes off. Even in cases which have advanced to true glaucoma we may usually distinguish between a permanent and a transitory element in the amblyopia. During each exacerbation which occurs in an inflammatory case of glaucoma, the vision is worse than before; after the acute symptoms subside there is a gradual recovery to a certain extent, which, however, does not as a rule lead to a restoration of the same degree of acuteness which existed before the attack. Each attack, therefore, leaves vision more impaired than before, until it is eventually lost altogether.

There is a very great difference in the rapidity with which complete destruction of vision takes place, a difference which depends on the nature and severity as well as the frequency of the acute attacks, and also upon the continuance of the increased tension. In the most acute cases, where the symptoms of congestion are excessive, vision may be altogether lost in a few hours. To such cases the name *glaucoma fulminans* has been given, while, on the other hand, chronic cases, and more particularly chronic non-congestive cases, may not lead to the complete loss of vision until after the lapse of many years.

These differences render it probable that there are two ways in which the vision is lost—(1) as the result of the increased tension within the eye, and (2) where the amaurosis is rapidly produced as the direct result as well of the vascular state which gives rise to the increased tension. Probably the suddenness and possible completeness of the ischæmia of the retinal vessels is sufficient to permanently abolish vision, just as it does in cases of embolism of the central artery of the retina.

Various views are entertained as to how the increased intraocular tension causes the amblyopia. By some, for instance, it is supposed that the pressure upon the fibres in the optic nerve directly leads to the blindness. Another hypothesis is, that the increased pressure on the retina is the cause ; while others, again, believe that the functional activity of the retina is gradually lost owing to an interference caused by the pressure in the blood supply to the delicate structures of the retina.

Restriction of the field of vision occurs at the same time as the defect in the acuity of the central vision. This restriction seems first to have been pointed out by Mackenzie. There is no absolute constancy in the manner in which the field of vision is invaded, but most frequently one finds the nasal portion abolished to a greater extent at first than other portions of the field, and not infrequently this portion alone can be demonstrated to be defective. Wherever the restriction is it is generally continuous; that is to say, there are not, as a rule, scotomata, but if one part of the retina has lost its function, that of the parts more peripheral to it in the same direction is mostly also abolished. The blind portion of the field of vision is bordered by an amblyopic area in which colour vision is either absent or defective, but there is not the same recession of the boundaries for colour vision as is met with in atrophy of the optic nerve. The limit at which the different colours are recognisable is usually much the same for all, and only slightly more retracted than the corresponding boundary for uncoloured impressions, while even in cases where the limitation is very great there still remains colour perception. It is the rule for this greater restriction to the nasal side of the field to progress and eventually involve the centre before complete blindness sets in, so that at an advanced state of the disease only an eccentric portion of the temporal side remains. The preponderance of the nasal invasion of the field appears to be more common in chronic than in acute cases of glaucoma. The manner of restriction next most frequently met with is the concentric restriction, where there is a tolerably proportionate interference with the function of all parts of the periphery of the retina. It is very rare indeed to find the restriction most marked outwards, and still more so, as some have seen, for this portion to be the only one in which the field is diminished. In not a few cases the central vision is markedly diminished without any easily demonstrable defect in the field while the opposite condition,

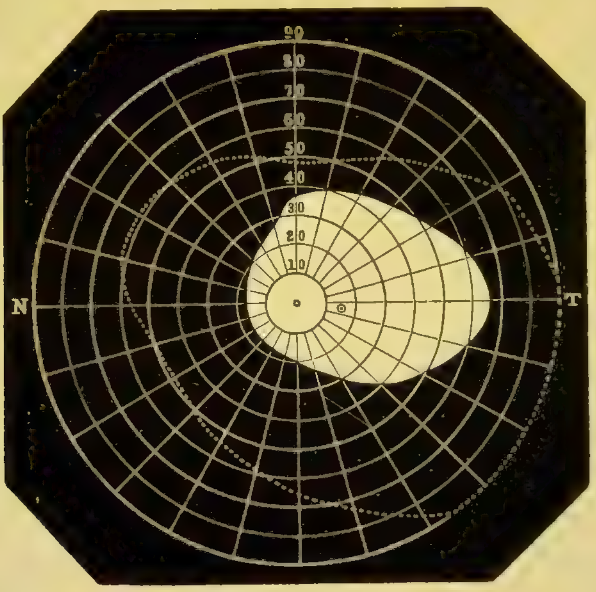

FIG. 99.—Typical defect of field of vision, from a case of glaucoma.

viz., great restriction of the field with good central vision, is rare, if it ever does occur, in pure glaucoma.

From an examination of one hundred cases, in which a glaucomatous excavation of the papilla existed, Bunge found the following forms of defect of the field of vision :—Defect in nasal portion alone, twenty-seven cases; predominating in nasal portion, forty-four cases; field remaining, assuming form of peripapillary oval, four cases; destruction of whole field, including centre, with exception of small temporal portion, nine cases. A central or paracentral scotoma, with or without slight restriction of nasal periphery, four cases; restriction only upwards, two cases; concentric restriction, six cases; preponderance of defect in the temporal half of the field, four cases.

Bjerrum has shown that if the glaucoma field be taken for a white object 3 mm. in diameter at 2 metres from the eye, and therefore for an object subtending a very small visual angle, there is almost invariably to be found a very characteristic form of restriction. The shape of the field is by no means always the same in all cases, but the defective area is always found to extend right up to the blind spot in one direction or another. In other words, the blind area corresponding to the entrance of the optic nerve is continuous in some direction with the defect in the field caused by the glaucoma, while in other directions it is found to have its normal boundaries.

In a number of cases of glaucoma which I examined with the assistance of Dr. Ramage, with reference to this point, we found Bjerrum's results fully confirmed. Fig. 100 is a drawing of a glaucoma field taken by Bjerrum's method. In several cases where I had performed iridectomy for glaucoma in the one eye, but in which no other evidence of glaucoma was present in the other eye, the same characteristic restriction was found to exist.

Bjerrum's symptom may then be looked upon as one of the earliest recognisable changes in most cases of glaucoma. The peculiar form of restriction described is not met with in any other disease.

A satisfactory explanation of the way in which the blindness is produced must account for (1.) the want of constancy in the manner in which peripheral vision is lost, and (2.) the tendency at the same time for the nasal portion to disappear before the other equally peripheral parts. The difference which is met with in respect to the function of the retina in acute and chronic cases respectively, seems to point to an absence of any likelihood of the cause of the amblyopia being always the same. Of the three hypotheses already mentioned, the one which ascribes the blindness to direct pressure on the retina appears to be the least consistent with the clinical facts. It was supposed by Donders that the reason why such pressure should affect the periphery in the first place, is that the nerve fibres which supply these regions are the most superficial. On this supposition it is difficult to understand why the manner in which the field of vision is invaded should not be more regular than it is, even taking it for granted—although it has not been proved, and appears most unlikely—that a pressure, exerted in the way the fluid pressure within the eye is exerted, would have the effect of completely and permanently interfering with the function of the more superficially coursing fibres, while that of the deeper ones remained relatively undisturbed.

As to direct pressure on the nerve, Nettleship, Bunge, and others have demonstrated that the fibres which supply the region of the macula occupy the greater part of the outer quadrant of the optic nerve as it enters the eye. There is much to be said, too, in favour of Bunge's views as to the position in the nerve of the fibres passing to other parts of the retina, viz., that the more centrally situated fibres supply the more peripheral portions of the retina. According to his scheme, an excavation occupying more specially and primarily one portion of the disc might possibly explain the visual defects, both central and peripheral, which are most commonly found to exist, if we were to assume at the same time that the fibres contained in the excavated portion of the disc were those whose function was most interfered with. Individual differences might also be assumed to

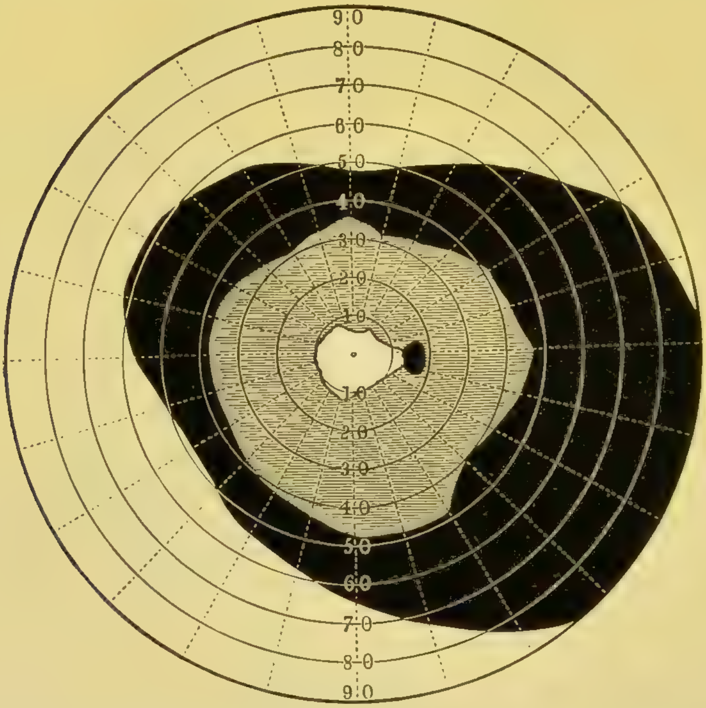

FIG. 100.—Field of vision from a case of glaucoma in which $V = \frac{20}{30}$. Shaded area marks extent of field by ordinary perimeter test; white area field for small object by Bjerrum's method.

exist with respect to the portions of the nerve which yield to the greatest extent to pressure, and thus account for differences in the visual defects. The assumption of a direct pressure on the fibres of the optic nerve, and their degeneration as the lamina cribrosa yields to the increased fluid pressure, is open to the same objection as has just been urged against the hypothesis of direct pressure on the retinal nerve fibres, viz., that it does not altogether account for the much greater destruction of function in some parts of the retina than in others, whose nerve supply in the papilla is not very far removed from the first. Besides, when there is complete loss of a portion of the nasal periphery of the field, there ought at the same time, were the pressure of the fibres in the papilla the only cause of this, to be a blind portion somewhere near the macula, or a paracentral scotoma. Such a scotoma is rare, however. It was only found four times in the hundred cases cited by Bunge.

The interference in function resulting from pressure on the retinal vessels is a factor to which there is little doubt some importance must be ascribed. The pressure may be supposed to act most effectually on the smaller terminal branches of these vessels, both directly and by compressing the larger trunks in the papilla, or it might be as a consequence of the kinking of the vessels at the margin of the excavated disc.

The more abnormal course of the amblyopia, as well as the blindness produced in very acute cases before any excavation of the papilla takes place, points to some factor independent altogether of pressure on the nerve elements at all events, and connected in all probability more directly with the vascular changes of which the increased intraocular tension is but one of the symptoms.

Bjerrum's symptom is certainly very suggestive of the amblyopia being due, in the majority of cases, to some change in the nerve itself. It does not, however, throw much light upon the question as to whether that change is due directly to pressure or not.

Photopsia, or the subjective sensation of light, is not an invariable symptom, and is besides a very inconstant one in any particular case of glaucoma. It is seldom very marked, but is sometimes present to a painful extent, and often persists even after the disease has led to complete blindness. Even at this time it is a variable symptom, being only experienced during the attacks which give rise to pain and increased tension. The cause of these sensations is in all probability some dragging of the retina which may be sufficient to give rise to a sensation of light, even at a time when the atrophic process has so far advanced as to have abolished all possibility of a response to the normal stimulus. It is a very common thing to hear patients who are absolutely blind from glaucoma declare that they can see light, and to find on examination no cause for this other than a mechanical one.

More or less *anæsthesia of the cornea* is met with in almost all cases of confirmed glaucoma. The anæsthesia is not as a rule equally marked over the whole extent of the cornea, but it is seldom that on careful exploration some point or points of diminished sensibility are not to be found. The test is best made by touching the cornea with a small feather or camel hair brush. The cause of this anæsthesia was generally believed to be the pressure on the ciliary nerve within the eye, until Fuchs advanced the more probable explanation of an interference by pressure or by a separation of the nerve filaments in the cornea as a result of the œdema, which is the anatomical change giving rise to the haziness of the cornea.

Fuchs explains the process which leads to anæsthesia of the cornea as follows :—" An increased amount of fluid finds its way through the nerve channels in Bowman's membrane, and thus causes their distension. The nerve fibres, which are surrounded and infiltrated, and probably also pressed on, by this fluid, are paralysed. At places where bleb-shaped elevations of epithelium are met with the fibres are actually torn asunder. When a false membrane of coagulated fluid has been found between the epithelium and Bowman's membrane the rupture of the nerve twigs takes place over almost the whole extent of the cornea."

Pain, varying in amount from a disagreeable sensation of fulness and dragging in the eye, to the most acute neuralgia over the whole region supplied by the fifth nerve, is a symptom which always marks at any rate the exacerbations in congestive glaucoma, and which in many cases is pretty constant. Unfortunately those who suffer from glaucoma do not, as a rule, get rid of the pain after the glaucoma has become absolute.

In very acute cases the irritation of the fifth nerve brings on somewhat alarming general symptoms, feverishness, sickness, and vomiting, which may be so severe as to detract attention from the local mischief, if one is not alive to the connection which may exist between the local cause and the general symptoms, and this all the more so, as even the patient may ascribe more importance to the latter. The pain, when moderate, is also due in most instances to irritation of the fibres of the fifth nerve supplying the eye ; but it is possible that sometimes at any rate an irritation of the fifth nerve is the primary cause of both the glaucoma and the other symptoms complained of.

The most common form in which glaucoma presents itself is as subacute attacks, following each other at intervals, the first beginning as a rule after some premonitory symptoms have previously been observed. After each attack of this nature a partial recovery of vision takes place, but the ultimate amount of vision is almost always less than it was before, the field more restricted, and the excavation of the papilla more marked. The exacerbations become eventually more frequent and permanent, and the recovery after each attack is less complete, until eventually the chronic congestive type of the disease is fully developed, and presents all or most of the objective and subjective symptoms already described. Even after a long continuance of the simple form of the disease, it is possible for acute symptoms to develop all at once. As a rule, however, cases of glaucoma simplex retain their character to the end. Probably many years may pass before the excavation of the nerve becomes complete; but when the case is first seen, there is generally marked cupping, although there may be no external evidence of any impeded circulation.

In a severe acute, so-called inflammatory, attack the circulatory disturbance leads not only to rapid and extreme increase of intraocular tension, but also to exudations into the external tissues of the eye, giving rise to chemosis, and even redness and swelling of the lids. The corneal opacity, too, is very great, the pupil dilated and absolutely immobile, while the severity of the attack is evidenced by the photophobia, lachrymation, pain vomiting, and general disturbance to which it gives rise. The vision, too, is either rapidly altogether lost or very much impaired. A subacute attack, on the other hand, is characterised by a less sudden deterioration as well as by a greater subsequent improvement of vision.

Acute or subacute attacks of glaucoma come on in eyes predisposed to the disease after the occurrence of anything which may be supposed to lead to a venous stasis in the uveal tract, either directly or owing to diminished arterial tension. Fits of mental depression and sleeplessness are thus likely to be followed by glaucoma, and this is also the case where there is a weakness of the heart's action, as during convalescence from illness. In short, any debilitating condition may be followed by glaucoma. Under such circumstances the pupil is apt to become dilated. This condition is of itself likely to favour the outbreak of glaucoma when the predisposition exists. Thus one sees every now and then an attack follow the use of atropine. Even homatropine and cocaïne have been observed to produce the same effect. On the other hand, a natural or induced contraction of the pupil when this can take place often cuts short an attack. This is no doubt one reason why sleep is found to be so beneficial in the early stage of the disease. As to the *predisposition*, it is seldom, unless premonitory symptoms have been complained of, that one can be alive to its existence. The only circumstance which would raise a suspicion in this respect would be the discovery of a greater degree of presbyopia than corresponded to the patient's age.

Diagnosis.—It is by no means always easy to diagnose glaucoma, owing to the variety of symptoms which in different cases are most prominent, and the absence often at the time of examination of one or more of the most important of these symptoms. It is unquestionably, however, the duty of every medical man in general practice to acquire such a knowledge of the clinical aspects of glaucoma, that he may be in a position to suspect, even if he may not be able to diagnose, its existence. The diagnosis is most difficult in the case of the simple form of the disease, and here sometimes the difficulty is very great. In all cases the form of excavation of the papilla, the presence of arterial pulsation, more or less irregularity of the pupil, fulness of the episcleral veins, anæsthesia of the cornea, and the condition of the colour and light senses, are the points to which attention must be given, or which must be looked for. It must be remembered that an evident increase of tension may be absent at the time of examination.

It is with some forms of atrophy of the optic nerve that one is apt to confound glaucoma simplex, and further, there is often great difficulty, where the glaucoma exists along with myopia from choroidal disease, in satisfying one's-self as to its presence. When the data afforded by the objective appearances are not sufficient to justify a diagnosis, and when no very characteristic restriction of the field of vision exists, some help may be obtained by an examination into the state of the light sense, and the peripheral colour sense. In cases of pure atrophy, *i.e.*, such in which the nerve elements of the retina are primarily involved in the process which gives rise to the amblyopia, it is found that while very little more amblyopia is caused by reducing the illumination, and the minimum perceptible amount of light is not markedly less than normal, a very distinct defect exists in the ability to distinguish between different intensities of illumination. On the other hand, the choroido-retinal affections exhibit exactly the opposite conditions with respect to these two elements of the light sense, viz., more or less marked night blindness, with relatively good power of distinguishing between different intensities of illumination. In glaucoma the defects in the light sense occupy an intermediate position—a fact which is not without importance in connection with the views as to the etiology of the disease, and which at the same time is of some diagnostic importance, although the practical value in this respect is hardly as great as has been supposed, owing to there being considerable differences in different cases in the nature of the defects of the light sense. Peripheral colour vision is not, as we have seen, so markedly defective in glaucoma as in atrophy, and the functional activity of the retina in this respect should consequently be determined in all doubtful cases.

SECONDARY GLAUCOMA.—Much the same set of symptoms, and occurring with different degrees of severity, are met with in eyes which are the site of other affections, either inflammatory or traumatic. There is then said to be secondary glaucoma, as the glaucomatous symptoms have not originated of themselves, but have followed upon some change which of itself is recognised as pathological. When the condition of secondary glaucoma is set up, the risk which the eye runs is the same as when the disease is primary, with this important difference, however, that the cause of the circulatory disturbance being apparent, of a more or less distinctly mechanical nature, and, as it were, less intimately associated with the symptoms to which it gives rise, there is, when it can be removed, considerably more chance of averting the consequences of the glaucomatous attack than is given by interference in the case of primary glaucoma. Thus glaucoma, along with iritis or traumatic cataract, can generally be arrested by operation. This is not the case, however, when the cause is a dislocated lens, or a dense leucoma adherens, leading to staphyloma, as the symptoms can in such cases seldom be removed by the performance of iridectomy.

One form of glaucoma, generally considered as belonging to the secondary category, though more correctly speaking primary, deserves special attention, viz., what is called *hæmorrhagic glaucoma.* In this disease glaucomatous symptoms of varying intensity supervene, generally after the lapse of some weeks, upon an attack of apoplexy of the retina. The pain is usually very great in such cases, and the condition may occur in one or both eyes, though fortunately more frequently in one alone. There is often a history of sudden blindness coming on before the attack of the glaucoma. The iris may present the appearance of hæmorrhagic infiltration, or there may be blood in the anterior chamber. The chamber itself is never shallowed in the manner so characteristic of other forms of primary glaucoma. The individuals in whom hæmorrhagic glaucoma occurs are almost always advanced in life, and the subjects of atheromatous degeneration of vessels. They not infrequently die of cerebral apoplexy.

A pretty distinct group of cases complicated by increased intraocular tension and other symptoms of a glaucomatous state, are those in which for some cause or other there is an abnormal degree of exudation passing from the iris or ciliary processes, as in serous iritis and where anterior synechiæ give rise to a distinct dragging on the iris, as may be the case in pretty extensive adhesions to linear scars, or to staphylomatous cicatricial tissue in the cornea. Other forms of iritis may become complicated by secondary glaucoma probably from another cause, viz., an interference with the escape of the aqueous fluid from the posterior into the anterior aqueous chamber which results from the existence of total posterior synechiæ; and when secondary glaucoma, owing to this condition, is present, one sees a more or less marked bulging forward of the iris.

Traumatic cataract, whether produced accidentally or by operation, is not infrequently followed by increased tension. Two conditions appear to give rise to this, viz.—the swelling of the lens substance within its capsule and the filling of the anterior chamber with the broken-up portions of the lens. The latter is more likely to cause this complication the greater the mass of lens matter occupying the anterior chamber, and the older the individual in whom the trauma has occurred. No doubt, too, some cataracts, both traumatic and congenital, contain substances which are more liable to set up irritation and induce tension than others.

Dislocation of the lens is another cause of secondary glaucoma. Intraocular tumours almost invariably, when they have attained any size, give rise to glaucomatous symptoms, which are sometimes so severe as to render the diagnosis of the primary disease very difficult owing to the haziness of the dioptric media. The more rapidly growing tumours are more likely to be complicated by secondary glaucoma than those which increase slowly and permit of the eye accommodating itself to the altered conditions, or of undergoing gradual degenerative changes.

Whilst primary glaucoma is almost exclusively a disease occurring late in life, secondary glaucoma is met with at all ages, although undoubtedly more is required to set it up in young eyes than in old. The vision, too, does not appear to be so rapidly or permanently interfered with by the increased tension in young eyes.

Two important forms of complicated glaucoma occur—(1.) cataract with glaucoma; (2.) high myopia with glaucoma. Both conditions are fortunately rare. With regard to the first, it appears almost as if the senile changes which brought about cataract created an immunity against glaucoma. But exceptions do occur, and it is important to diagnose the existence of the glaucoma, as special care is required in the treatment.

Most frequently it would appear that such a complication is confined to one eye; at least it is not such an uncommon thing to meet with senile cataract at the same time in both eyes, and glaucoma in the one alone. It is possible that such cases come as a rule under observation much sooner after the blindness of the second eye, and the operation for removing the cataract has a prophylactic action on the glaucoma, to which there may have been a tendency.

Sometimes one meets with glaucoma after extraction, *i.e.*, in an aphakic eye. Possibly in such cases the conditions likely to be followed by an outbreak of glaucoma existed previous to the operation; but it is more likely that, as a rule at any rate, the glaucoma is secondary, and caused by the dragging on the peripheral portion of the coloboma made in the iris, or on a portion of the lens capsule, which is incorporated with the cicatrix. Another cause may be the exudation set up by the dragging on the iris or ciliary body of the contracting elements, which make up the more or less opaque secondary cataract, as needling of this membrane is sometimes sufficient to check the glaucomatous symptoms. When a chronic congestive glaucoma has reached the stage of degeneration, the lens not infrequently becomes opaque. Such cases are, however, not to be confounded with the group of complicated glaucomas just referred to.

Whether or not glaucoma along with senile cataract is to be looked upon as a secondary glaucoma, or as an independent coincidence of the two diseases, there can be little doubt that glaucoma in a highly myopic eye is, properly speaking, secondary, inasmuch as the choroidal changes which lead to the elongation of the globe probably interfere at the same time with the circulation in that membrane, and lead to a glaucomatous complication. From a clinical point of view, it is better to look upon the form as a complicated glaucoma. The tension is rarely very high, though above normal; the field of vision becomes restricted in a manner more or less characteristic of glaucoma, and the papilla is excavated. It is the excavation of the papilla, as well as sometimes the unusual amount of pain complained of, which should lead one to suspect this complication. The excavation is never so deep as in other forms of glaucoma, but it is unmistakable whenever it is seen to reach out to the margin of the papilla. In these cases there is always a very marked and often excessive degree of choroidal atrophy surrounding the papilla, and it is generally supposed to be owing to the comparative want of resistance in the surrounding parts that the yielding of the lamina cribrosa does not assert itself to the same extent as would otherwise be the case.

Prognosis.—The prognosis in glaucoma, in whatever form the disease may present itself, is always bad whenever the glaucoma is confirmed, *i.e.*, when it has begun to produce an impairment of vision. The premonitory stage may exist for a long time, and may even never lead to confirmed glaucoma. It is rare, if indeed it ever happens, that the disease becomes spontaneously arrested, so that, for practical purposes, we must consider that glaucoma left to take its own course invariably leads to total destruction of vision. The time taken to this work of destruction varies within enormously wide limits,—from a few minutes to many years.

When an operation is performed, the prognosis may be said to depend on the stage of the disease at which it is undertaken, on the nature of the operation, on the manner in which the operation has been performed, and the way in which healing has taken place. Not a little depends, too, on the form of disease treated, and the presence or absence of complications.

The *treatment* of glaucoma should differ according to the stage of the disease. Where only occasional premonitory symptoms are present, and vision is therefore good during the intervals, there may be a reluctance on the part of the patient to undergo an operation. The local application of eserine may then be adopted. A half per cent. solution dropped into the conjunctival sac speedily cuts short these attacks. At the same time attention should be directed to the general health, and if the heart's action be weak, iron and digitalis prescribed. A properly performed iridectomy at this stage not only checks the premonitory attacks, but almost certainly prevents the progress of the disease altogether. There should, therefore, be no hesitation in proposing an iridectomy even at the early stage, and all the more urgently if the symptoms frequently make their appearance, as it is impossible to say how much damage may be done, and done irretrievably, as soon as the disease becomes confirmed. Unquestionably, however, as soon as the glaucoma has begun to interfere permanently with the function of the eye an iridectomy should be performed. The method of performing this operation, and the special precautions to be taken in the case of glaucoma, are given in Chapter XVIII. Iridectomy is the only cure for glaucoma, and is the more likely to be successful the more the iris tissue has retained its normal character. When atrophic changes have begun, much depends upon their extent, whether removal of a portion of iris will be sufficient or not to permanently arrest the glaucoma. It may be taken as a rule that the further advanced the process, the larger should be the portion of iris removed. In all cases the iris should be cut as peripherally as possible, but should not be detached from its ciliary attachment. Great care should be taken to prevent any portion of the iris becoming entangled in the wound. This, which is called encleisis of the iris, is very apt to interfere with the effect of the operation, as the iris is not only dragged upon, but prevents the firm close healing of the external wound which should be aimed at.

The first effect of an iridectomy for glaucoma is generally to reduce the tension of the eye. Where it does not do so, the prognosis is mostly, though by no means always, unfavourable. The reduction of the tension very soon relieves the pain which may have existed. In acute cases, where the cornea has been very hazy, the rapid disappearance of the œdema, to which the haziness is due admits of considerable improvement of vision, though this may be marred by blood effused from the cut surface of the iris. Bleeding from the iris is not uncommon, and may take place at the time of the operation, or several days afterwards.

In acute cases there may be added to the improvement of vision, due to the clearing of the cornea, a further improvement which results from the circulation in the uveal tract assuming a more normal character. Two or three weeks, or even longer, generally elapse before the restoration of vision has reached the full amount possible. In subacute and chronic cases the same amount of improvement of vision is not to be expected, and the operation will have served its purpose if the vision existing at the time of its performance is retained. Often, indeed, in the case of simple glaucoma, iridectomy, although arresting the progress of the disease, leads immediately to some deterioration of vision, and this is more especially evident in the peripheral vision, the field of vision being not infrequently found to be narrower after than immediately before operation. It is well to remember this, as it sometimes happens that in cases where the nasal portion of the field is restricted to nearly the point of fixation, that point becomes itself included in the blinded area by the slight increase in the restriction following the operation, and the vision thereby rendered all at once very much worse. In more acute cases of glaucoma, the previous use of a myotic not only facilitates the performance of the iridectomy, but renders the accidents which may occur on the sudden reduction of the tension less liable. It is well, too, in operating for acute glaucoma in one eye, when the other has so far exhibited no signs of the disease, to follow the advice given by Arlt, and keep the sound one under the influence of a myotic, as the mental anxiety of having to submit to an operation has not infrequently been observed to induce an attack of glaucoma in the hitherto sound one. Arlt did not meet with this unfortunate complication after adopting the treatment of using eserine for the sound eye.

Where glaucoma exists along with cataract, it is comparatively seldom that it is of any use to extract the lens, as vision has generally been already lost. Where, however, an extraction is indicated, it is probably best to perform iridectomy some weeks previously. In the true hæmorrhagic glaucoma, iridectomy almost certainly leads to further hæmorrhage and rapid destruction of vision. In such cases, therefore, the operation is on the whole contra-indicated.

In some of the cases of simple glaucoma in which the tension is not diminished immediately after the operation, but the eye remains stone hard, a rapid loss of sight takes place. To these cases the name of *malignant glaucoma* has been given. Sometimes the cause of this is a subretinal hæmorrhage. Usually, however, there is a forward displacement of the lens leading to a blocking of the channels for the escape of fluids at the angle of the anterior chamber. In these cases the proper treatment is to extract the lens.

Even when iridectomy fails to arrest the progress of the blindness, it generally protects the eye against the occurrence or recurrence of a congestive attack of glaucoma. Iridectomy is not only capable of reducing tension, but of regulating it to a certain extent, so that, when performed for cases of chronic irido-cyclitis, in which diminution of tension has taken place, it often leads to a more normal tension; therefore instead of reducing, it actually increases the hardness of the eye. Whatever be the nature, then, of the curative action of iridectomy in glaucoma, it is certain that it does more than merely reduce tension. In some way or other it also induces a more normal condition of the circulation in the uveal tract. The manner in which this is supposed to be effected is discussed under the etiology of glaucoma.

Iridectomy as a cure for glaucoma was introduced by von Graefe in 1857. He was led to the discovery purely empirically, but at the same time as the result of great clinical experience and extraordinary insight, and thus conferred one of the greatest blessings on humanity, and made the greatest advance in ophthalmic surgery of modern times. Attempts have been made lately to detract from the merits of von Graefe in this connection. The injustice of such attempts will be apparent to any who has read the literature of the subject of that time, and especially the writings of von Graefe himself in the years 1855, 1856, and 1857.

The statistics of Horner (as given by Sulzer) from 1861 to 1881 are as follows:—They are based upon 103 iridectomies for simple glaucoma, and 149 for inflammatory glaucoma.

	Infl. Glaucoma.	Simple Glaucoma.
Improved, . .	72·5 per cent.	22·3 per cent.
As before operation,	11·5 ,,	37·0 ,,
Partly retained, .	10·0 ,,	23·0 ,,
Not improved, and ultimately lost, . .	6·0 ,,	17·7 ,,

On the supposition that iridectomy merely acts as a reducer of tension, and thus cures glaucoma, another operation has of late years been substituted for it, viz., sclerotomy. The different methods of performing this operation are described in Chapter XVIII. There is not the slightest doubt that the efficacy of sclerotomy is greatly less than that of iridectomy. In most cases it is of no use at all, but it does appear to be occasionally capable of arresting the disease.

There are some cases in which sclerotomy may with advantage take the place of iridectomy. Cases of hæmorrhagic glaucoma, for instance, seem to be less liable to go wrong if treated by sclerotomy instead of iridectomy; and in all cases where iridectomy has been followed by bad results in the first eye, which cannot be ascribed to any fault in the operation, it is well to resort to sclerotomy in the second. It is a question, too, whether, in the worst of such cases at least, sclerotomy is not the more preferable operation in myopia complicated with glaucoma, in which the prognosis is in any case rather hopeless.

Other operative measures in use for the treatment of glaucoma are:—Paracentesis of the anterior chamber; division of the ciliary muscle, or cyclotomy; and trephining of the sclera. Paracentesis sometimes reduces the tension, and is resorted to as a preparation for iridectomy; and in cases where the cornea is hazy it is followed by a certain amount of improvement of vision. By some it is pretty extensively used, not only as a preliminary operation, but as a means of promoting the efficiency of iridectomy by keeping down, as they believe, any tendency to increased tension during the time the healing is taking place. Cyclotomy is rarely practised. It is capable of reducing tension, but is by no means a safe operation. The operation of trephining the sclera has been advocated by Argyll Robertson as a last resource in cases in which iridectomy fails to reduce tension at all. Notwithstanding the risky nature of the operation the results have been encouraging in suitable cases.

Etiology of Glaucoma.—Although the different forms of glaucoma are grouped together as one disease, it seems hardly probable that the main symptoms which they present in common, viz., increased intra-ocular tension and a more or less gradual deterioration in the functions of the retina, are in all cases the expression of one and the same pathological process.

The glaucomatous eye retains the increased hardness which it may have had for some time after excision, so that there cannot be any doubt that the increased tension is the result of an actual abnormal fluid over distension of its coats. This being the case, the question for solution is—Is the increased distension due to increased exudation of fluid into the eye, or to an impeded outflow of a normal amount of exudation, or to both, or in some cases to one or other alone? A satisfactory answer to this question must also account for the relative rarity of the disease in individuals under a certain age.

One of the great difficulties experienced in connection with the pathological anatomy of glaucoma is to determine what changes are primary and what secondary, as the great mass of material which can be utilised in this way consists of eyes enucleated in a late stage of the disease, when more or less atrophic changes have taken place. And when one takes into consideration the long-continued existence of vascular changes in the eye, it is not strange that all parts of it should have been found to exhibit pathological appearances, to some one or other of which primary importance has been attached by different investigators. Knies, for instance, found, as had long before been found by H. Müller (who, however, did not attach any importance to it), that an obliteration of the space of Fontana, at the angle of the anterior chamber, was constantly present in all cases of glaucoma which he examined. He found the whole surrounding of Schlemm's canal infiltrated with cells in fresh cases, whereas in old ones a cicatricial contraction of this inflammatory exudation had taken place concentrically towards that channel for the escape of fluid from the chamber. It was noticed, further, that whilst in chronic cases the inflammatory changes were limited to this area, in acute cases the ciliary body, iris, and subconjunctival tissue participated in them. According to Knies, it is not extraordinary that a situation so functionally active as the space of Fontana should be subject to an independent inflammation, and further, he holds that its obliteration must necessarily lead to an increase in the intraocular tension. On the assumption that such changes are the cause of glaucoma, he believes he can explain in this way the opacity and anæsthesia of the cornea, the irido-plegia, apparent shallowing of the anterior chamber, the paresis of accommodation, and the enlargement of the episcleral veins, which characterise the glaucomatous state. He even goes so far as to assert that the constancy of the last symptom, compared with the inconstancy of the others in different cases, is a point of the utmost importance in support of his views on the nature of glaucoma. While he admits that it is open to discussion whether or not the primary inflammation of the vicinity of Schlemm's canal is to be looked upon as the sole cause of glaucoma, he maintains that it is of fundamental importance in the production of many of the symptoms, and must be looked upon as the most important pathological change after the excavation of the optic nerve.

This certainly very significant discovery was not long in being shown to be inapplicable as an explanation for all cases of glaucoma. Both Brailey and Pagenstecher, for instance, found that there were cases of glaucoma in which no obliteration of the space of Fontana exists. Other conditions than glaucoma have been found, too, to be associated with obliteration in this situation. The natural inference is of course that the appearances described by Knies, when they do exist, are secondary appearances, and not the primary cause of the glaucoma, and in fact it has been pretty well shown that adhesion of the peripheral portion of the iris to the cornea occurs frequently where, for some cause or other, the two surfaces are brought into contact for any length of time. At the same time, although this condition is frequently associated with glaucomatous symptoms, there is no necessary connection between the two.

In the belief that the fluids of the eye mainly escape by the known anterior lymph channels, Priestley Smith has put forward an hypothesis to account for increased tension. It is evident that if there be no primary blocking of the space of Fontana and Schlemm's canal, and yet these channels afford the only means of escape for fluids from the eye, the advance of the iris and lens would only be likely to take place if some impediment existed somewhere to the passage forwards of fluids from the vitreous. Priestley Smith considers that such an impediment is offered by the more or less complete obliteration of the circumlental space which separates the margin of the lens from the ciliary processes. A prolonged investigation has led him to the interesting discovery that this space gradually diminishes in size as age advances, owing to the constant increase which takes place throughout life in the size of the lens; so that in advanced life any little swelling of the ciliary processes would obliterate it completely. According to Priestley Smith, then, the order of occurrences is, first, the choking of the circumlental space by a hyperæmic swelling of the ciliary processes, then, the channel of communication between the two chambers being obliterated, fluid begins to accumulate in the vitreous, and the lens and iris, more especially the latter, are pushed forward, the angle of the anterior chamber thus blocked, and a vicious circle established, by which more or less rapid increase of intraocular tension is not only produced but maintained, with all the accompanying symptoms of the glaucomatous state. Much the same explanation has also been given by Weber, who claims to have produced a condition in rabbits resembling, both clinically and anatomically, glaucoma simplex, by injecting oil into the anterior chamber. Priestley Smith, therefore, with others who do not admit the existence, as a rule at all events, of primary inflammatory changes at the angle of the anterior chamber, look upon the blocking of this important region of the eye as secondary and produced by compression, though they maintain that only when this blocking takes place are the conditions fulfilled which lead to a rise in the tension of the eye.

Priestley Smith's explanation accords better with clinical facts in ascribing the primary cause to venous stasis in, and consequent swelling of, the ciliary processes. The intermittent character of the increased pressure in so many cases is of itself more suggestive of active exudation than of accumulation of fluids in the eye due to interference with their escape. The question naturally arises, what evidence have we of any circulatory disturbance in glaucoma such as would lead to an increased secretion of intraocular fluids? It must be admitted that there is no very conclusive evidence from an anatomical point of view. As has already been remarked, however, changes found in glaucomatous eyes can rarely with any certainty be looked upon as primary, and in some way connected with the outbreak of the symptoms of the glaucoma, and not rather secondary, or resulting from the prolonged inflammatory or atrophic changes met with in the disease. But if direct anatomical evidence is difficult to obtain, there are many clinical reasons for assuming that a stasis takes place in the circulation within the eye. Thus the greater tendency to the disease the older the individual, its occurrence along with evidences of atheromatous degenerations elsewhere, and the tendency that there is for an outbreak to take place after debilitating diseases, mental depression, and other circumstances giving rise to a slowing of the heart's action, are all suggestive of this. So also are the action of iridectomy and the subsequent atrophic changes seen in the iris. The fact that the improvement following an iridectomy is only gradual, points to the cause of the improvement being the gradual return to more normal conditions of circulation, while the œdema and the injection of the external vessels in the acute cases, and the permanent distension of these vessels sometimes met with in chronic cases, although they conclusively show that there is an interference in the intraocular circulation, do not of course prove that such has been the primary cause of the symptoms.

The great variations in the severity of the disease, as well as the occurrence of an onset of glaucoma in some individuals from conditions which in others would have no similar result, show that there are two elements in the production of the circulatory changes. The one element is no doubt a pathological one of some kind, and when alone active may account for the cases of glaucoma simplex. The other, which can only be of great moment when occurring in eyes in which the first is present, must be looked upon as vasomotor; at all events all the so-called exciting causes of the disease point to this, while the exact nature of the temporary changes produced can only be a matter of conjecture. In the most violent attacks the second element, or the venous stasis, may be supposed to predominate.

There are reasonable grounds for believing, then, that the external appearances of the glaucomatous condition, as well as the increased intraocular tension, are due to circulatory changes in the eye, although the exact nature of such changes, or of all the factors which in every case give rise to such changes, is not yet known. In how far circulatory changes are to be held responsible for the accompanying visual disturbance is another question. It is generally supposed that in some way or other the increased tension leads to the destruction of vision, and, as has already been said, different views are entertained as to whether this is by pressure on the nerve, on the retina, or on the vessels from which the retina derives its nourishment. Any of these views, however, seems to be insufficient to account for the cases in which vision is rapidly and permanently lost, and also for cases in which well-defined scotomata are met with in different parts of the field of vision. For such cases there must be some other explanation, and it appears not unlikely that, just as with the conditions giving rise to stasis in the anterior section of the eye, we must assume that there is a permanent as well as a transitory element in the process which leads to the loss of the functions of the retina. The element which is more or less permanent may be the pressure element, or the existence of diseased conditions which cause the nerve to yield, as it were, to pressure. Possibly, indeed, some changes take place in the retinal arteries which independently lead to blindness. Brailey has, for instance, described a thickening and hyaline degeneration of their walls in glaucoma, and the fact that hæmorrhages often take place from the retinal vessels immediately after iridectomy is suggestive in this respect. As to the more transitory element in the loss of vision, Schnabel makes the very suggestive assumption that it is due to a spasmodic contraction of the retinal arteries. As glaucomatous attacks come on under circumstances which render vasomotor changes very likely, it certainly cannot be said to be altogether inconsistent with clinical experience to assume such a constriction of the arteries of the retina, as well as those of the iris and ciliary body. It is unlikely that the retinal functions are lost from pressure alone, as the only way in which this could well be possible would be for the pressure to produce an almost complete stoppage in the circulation; while, if this is really what happens, it cannot be a diffused pressure all over the retina which would cause narrowing of both arteries and veins, but a pressure which acts mainly on the vessels as they enter the retina from the nerve, causing diminution in the size of the arteries, and more or less engorgement of the veins, corresponding to the appearance actually met with in glaucoma. The possibility of interference in the circulation taking place mainly in this way would depend either upon whether a certain degree of pressure would be more effectual in compressing the vessels in the nerve than elsewhere, or whether there really existed a greater degree of pressure in this particular direction,—neither of which conditions there are any reasons to assume actually exist. Although the structures against which the vessels would be compressed are probably no more resistant in the nerve than in the retina, the same degree of pressure might cause greater interference with the circulation when exerted on points where sudden bends take place in the vessels, as happens where they pass into the retina.

One of the great difficulties, which it cannot be said has yet received a satisfactory explanation, is to account for the curative action of iridectomy in glaucoma. The facts are as follows:—The operation is more efficient when performed at a time when the iris tissue has not undergone any marked atrophic changes. It is also more efficient if it be followed by a normal healing process—that is to say, if the resulting cicatrix is flat and almost invisible, and does not in any way bulge or entangle iris tissue at its corners. Apparently also the more peripheral the section of the iris can be made, the better is the effect, though any tearing of it from its attachment to the ciliary body is less likely to lead to a good result. Iridectomy, while capable of arresting both the acute and chronic forms of glaucoma, is generally supposed to be more effectual in the former. This may, however, possibly depend upon its being performed, as a rule, in an earlier stage relatively, where the symptoms are acute. The action of iridectomy in glaucoma appears to be intimately connected with, but not actually dependent upon, the influence which it has in permanently reducing the abnormal degree of intraocular tension. Another curious point in connection with the operation is that it appears, as von Graefe first pointed out, to be capable of causing a return to a normal state of tension in cases of chronic irido-cyclitis which have led to diminished tension. All that can be said, then, is that in some way or other, and by reason probably of the changes which the excision of a portion of iris produce in the circulation in the iris, the operation of iridectomy is capable of effecting an improvement in the conditions of circulation of the ciliary body and anterior portion of the choroid.

Glaucoma derives its name from a symptom which is neither constant nor entirely pathognomonic—viz., a greenish (γλαυκός) reflection from the transparent media of the eye. This appearance was known to Hippocrates, though he does not appear to have recognised it as associated with any distinct form of disease. Many of the older writers evidently confounded glaucoma with cataract, or at all events considered the lens to be the part of the eye affected. Brisseau, in the beginning of last century, demonstrated by *post-mortem* examination that this was not the case, and declared glaucoma to be due to disease or turbidity of the vitreous humour. This view was not generally adopted until it was revived a century later by Beer, the weight of whose authority brought it into prominence. Beer also pointed out the arthritic nature of the disease.

Mackenzie seems to have been the first to have recognised the importance with regard to vision of the increased hardness of the globe which is met with in glaucoma, and to attempt systematically some means of diminishing this intraocular tension. The method adopted by Mackenzie, viz., paracentesis of the sclera, did not lead to any encouraging results, and was consequently abandoned by him. This circumstance led him to attach less importance to the increased tension than he had at first done, so that it was not until after the invention of the ophthalmoscope, and when by its aid more light was thrown on the intraocular changes occurring in glaucoma, that Mackenzie's views were, so to speak, confirmed and extended by von Graefe, who also discovered the means of curing the disease. From a clinical point of view, we owe our present knowledge of glaucoma to von Graefe.

CHAPTER XII.

INTRAOCULAR TUMOURS.

Sarcoma of the Choroid and Ciliary Body.—Much of our present knowledge with reference to intraocular tumours is of very recent date. It was only, indeed, after Virchow had shown that a tumour is essentially characterised by its histological structure, and not by its greater or less degree of pigmentation, that any useful light was thrown on the subject. The combined efforts of Virchow and von Graefe very soon led to order in the previous confusion of ideas. In 1868 von Graefe added to the list of his great works such a complete clinical history of intraocular tumours that little remained afterwards to be done.

Sarcoma of the choroid is usually circumscribed, but occasionally occurs as a diffuse infiltration of the choroid. Such cases have been described by Schiess, Hirschberg, and Fuchs. The ordinary circumscribed tumour may or may not be distinctly pedunculated. It detaches the retina in front of itself, only rarely perforating it. Sarcoma of the choroid may be either pigmented or not. The pigmented cases are much more common.

According to the statistics collected by Fuchs, about nine cases of melano-sarcoma occur for every one of leuco-sarcoma. This is no doubt owing to the presence of pigment in the choroid, as the pigmented form only occurs where pigment is physiologically present. In many cases the pigment is not found all over the tumour, and in hardly any is the degree of pigmentation equal throughout. The histological arrangement of the pigment granules also varies considerably in different cases, and these granules may be of different degrees of fineness. The coarser they are the blacker do they make the tumour appear. The pigment, although, as its chemical reactions show, derived from the blood, is probably directly formed by proliferation of the uveal pigment. The distinction usually made between melano-sarcoma and leuco-sarcoma is a somewhat arbitrary one, as some really pigmented growths have not a very pigmented appearance, and the transition in colour, taking a whole series of cases, is found to be a very gradual one. From a histological point of view, a sarcoma is melanotic whenever it has given rise to proliferation of the uveal pigment, however slight that proliferation may be, but when very slight it is impossible to recognise this clinically.

Leuco-sarcoma occurs mainly in the anterior portion of the choroid, and is met with, on the whole, at an earlier age than the melanotic variety. Round and spindle-celled sarcomata are the most common. Many other varieties have been described, which are, however, mainly interesting from a histological point of view.

Of 259 cases of sarcoma met with by different observers, the records of which have been collected by Fuchs, sixteen, or six per cent., occurred in the iris, twenty-two, or nine per cent., in the ciliary body, and 221, or eighty-five per cent., in the choroid. Of the twenty-two occurring in the ciliary body twenty were pigmented. Of the choroidal cases 196 were pigmented, and twenty-five unpigmented.

The most common point of origin for sarcomata of the choroid is at or near the posterior pole of the eye, a little to the outer side of the disc. They take their origin from the layer of big vessels. After the tumour has grown to a certain size in the eye, it perforates the coats and extends outside of it. The sclera is most frequently perforated; sometimes also the cornea and the optic nerve. Perforation through the sclera takes place at the corneo-scleral margin or in the vicinity of the optic nerve, or sometimes at other parts where it is weakened by the passage of vessels through it into the eye. Often, owing to the resistance offered by the sclera, the band of connection between the intra- and extra-ocular portions of the tumour is very small, but more and more of the sclera is usually destroyed as the tumour increases in size, which it may do if left alone until it has attained very considerable dimensions—as large as the closed fist or even larger. Notwithstanding such an increase in size, the bones of the orbit are very rarely affected, though sometimes an extension of the tumour takes place along the optic nerve to the brain.

The lymph glands in the neighbourhood of the eye are never affected in cases of uveal sarcoma, which in this respect differs from both carcinoma and glioma. The tendency to metastasis is however, very great, so that in all probability it is through the blood that the tumour elements are carried to a distance. As Fuchs has shown, a great deal evidently depends upon whether in the primary tumour the conditions are or are not present which favour the separation and removal by the blood current of particles of the growth. The more vascular the tumour the thinner and larger its vessels, and the softer the new tissue the more likely is this to happen, and therefore the greater is the malignity of the growth.

When the sarcoma grows from the ciliary body it grows forward into the posterior or anterior aqueous chambers, as well as backwards into the vitreous. It may thus become visible on simple inspection of the eye before it attains any very great size. Often the extension to the anterior chamber, which takes place by perforation through the peripheral portion of the iris, is preceded by a bulging forward of the iris where it covers the tumour. A tumour in this situation occasionally sets up severe irido-cyclitis, by which the diagnosis may at first be rendered difficult. Tumours which take their origin farther back detach the retina in front of them. At first the portion detached lies immediately over the tumour, and it is often difficult from ophthalmoscopic examination alone to tell whether the detachment is simple or produced in this way. One point, viz., the absence of any tremulousness on the movement of the eye, awakes suspicion. The defect of the field of vision is, as a rule, where a choroidal tumour is the cause, much more sharply defined, and there may also be, when the site of the growth is peripheral, relatively better central vision than is generally to be found in simple detachment. The tension of the eye is also an important guide to the differential diagnosis. It is most frequently increased in the case of a tumour; most frequently diminished when the detachment is idiopathic. After the tumour has existed for some time and increased in size, the whole retina gradually becomes detached, and assumes the funnel or convolvulus shaped form which is characteristic of complete detachment. Thus further detachment is evidently the result of a serous effusion to which the irritation of the new growth gives rise. Whether this effusion takes place directly into the subretinal space or first passes into the vitreous chamber, and subsequently behind the retina after the dragging on it has led to rupture, has not been clearly made out.

As to the frequency of uveal sarcoma, I find that of 181,853 patients attending Moorfields Hospital, London, and the Manchester Eye Hospital during four and five years, eighty-two are recorded as being cases of this disease. This is a proportion of one to 2218. Fuchs collected ninety-one cases from a material of 137,545 patients attending the clinics of ten different ophthalmic surgeons in Germany. Their statistics show a greater frequency of one to 1500. It occurs about equally frequently in both sexes, and is for the most part a disease of advanced life. Only eleven out of 259 cases collected by Fuchs occurred before the age of ten, only twenty-seven before the age of twenty. It is very rarely bilateral. Five of Fuchs' cases, or two per cent., had it in both eyes.

It has been supposed that the disease is more common in darkly pigmented eyes, and in cases where there is an abnormal amount of pigment in other isolated parts of the body; but any definite predisposition arising from this or any other cause—such as, for instance, heredity—though it has often been suspected, has certainly never been clearly demonstrated as yet. It is more probable that some connection may exist between certain inflammatory conditions of, and injuries to, the choroid, and the tendency to tumour formation. Such a connection is, in my experience, undoubtedly present in the case of orbital growths.

The course of uveal sarcoma is generally, according to a plan first adopted by Knapp, divided into four stages:—(1) The quiescent or non-irritative stage; (2.) The irritative or inflammatory stage; (3.) The extraocular stage; and (4.) The metastatic stage.

According to the statistics already several times referred to, the average duration of the *first stage* is from eighteen to twenty-one months, the lower limit being for cases of sarcoma of the ciliary body, the higher for those of the choroid proper. In many cases, no doubt, the period intervening between the first appearance of the growth and the occurrence of irritation is longer, but escapes observation. The very beginning of the growth has only rarely been seen as a flat elevation from the surrounding fundus. Usually at the first examination a brownish or yellowish protuberance is seen, on which, when the retina is not too intransparent, an indistinct and irregular network of vessels may be made out lying behind the retinal vessels. Anteriorly situated growths may sometimes be made out very distinctly by oblique illumination, if the pupil be well dilated with atropine.

In the *second stage* the eye is painful, and the tension more or less markedly increased. Often the condition presents much the appearance of inflammatory glaucoma. From the idiopathic or primary form of that disease, it is recognised mainly by the absence of any periods of remission which constantly occur in true glaucoma. When set up by a tumour, the inflammation and increased tension is usually constant. The duration of the second stage is less than of the first—on the average not longer than a year, and often considerably less.

The *third stage* is then entered upon by the tumour perforating and growing externally. The growth then becomes very rapid as a rule, and ulceration, accompanied by a purulent and generally offensive discharge, takes place on the surface. The pain at this stage often considerably abates at first, but reappears as a rule when proptosis, owing to extension of the tumour to the orbit or lids, becomes marked. The duration of the second stage, and therefore of the period of painful increase, is generally less in tumours of the ciliary body than in those of the choroid, owing to the greater ease with which they cause perforation.

As to the *fourth and final stage*, all that can be determined with any certainty is the period at which metastasis, which most commonly occurs in the liver, reveals itself by the appearance of symptoms. The tumour growth may of course begin, and in many cases no doubt has begun, long before it gives rise to any symptoms.

Diagnosis.—The chief difficulty of diagnosis presenting itself during the first stage is to distinguish a choroidal sarcoma from idiopathic detachment of the retina. The points to be attended to in the differential diagnosis have already been explained. Detachment of the choroid might be mistaken for sarcoma, but apart from the very great rarity of that affection, the suddenness of onset and the diminished tension would be taken into consideration. Glioma of the retina ought not, in the first stage, to be mistaken for sarcoma, and could only possibly be mistaken for the non-pigmented variety, as the colour is very different from most cases of sarcoma. With the ophthalmoscope, too, the glioma can be distinctly made out to be in the retina itself, the structure of which is altered in the portion occupied by the tumour, and seen not to be merely pressed forward and evidently covering the growth, as is characteristic of an entirely choroidal tumour. In the second stage, especially if the media be clouded, the difficulty is greater, and the diagnosis will depend in great measure on the age of the patient. Glioma always occurs in the first decade of life, whereas we have seen that the proportion of the cases of sarcoma occurring so early in life is only about 11 to 259, or not much over four per cent. Diffuse tuberculosis of the choroid has been mistaken for sarcoma, and probably in these rare cases the differential diagnosis would be very difficult.

In the second stage of sarcoma a confusion with idiopathic inflammatory glaucoma might be made, although such a mistake ought to be uncommon if proper attention be paid to all the circumstances connected with each case. In glaucoma the vision has usually been good before inflammatory symptoms have set in; there have, besides, often been observed the characteristic premonitory symptoms. There is the constant tendency to remission in the pain, and in the increased intraocular tension, already referred to. When the glaucoma is absolute in the one eye, there are often symptoms of the disease already existing in the other. In the case of sarcoma, on the other hand, the patient has usually been conscious of more or less blindness of the eye before it becomes painful. Often the blindness has come in the form of a definite positive scotoma. When the inflammatory symptoms set in they are constant. A brightish reflection from the completely detached retina can often be seen. Both eyes are hardly ever affected at the same time. In cases where the choroidal tumour gives rise to iridocyclitis, the diagnosis may certainly be rendered difficult, and still more so when shrinking of the globe takes place as the result of this inflammation, as sometimes happens. In idiopathic iridocyclitis the sight has probably been good before the onset of the inflammation, and is only slowly lost as it progresses. It often, too, affects both eyes. The shrunken eye resulting from simple irido-choroiditis is usually painful on pressure, but seldom subject to any great spontaneous pain, which, on the other hand, characterises that which encloses a tumour. This was first pointed out by von Graefe, who also directed attention to the form of the shrunken sarcomatous globe, which is more flattened from before backwards than from side to side, so that the depression caused at the sites of the recti muscles are more apparent in the front of the globe than in the case of the more ordinary variety of phthisis bulbi.

The *prognosis* in uveal sarcoma, if an operation be not undertaken, is as bad as possible. The disease is probably invariably fatal, and the time the patient has to live from its first appearance is not much more than five years, and often considerably less.

Of the cases on which Fuchs founded his statistics, 285 were operated on, and of these 31, or thirteen per cent., were followed by local recurrence of the growth. In most of the cases recurrence took place within a year. The statistics show also what must be looked upon as an important point practically, that if recurrence does not take place within four years after removal of the eye it is not likely ever to make its appearance. Occasionally exceptions do occur to this rule; thus in one case of Hirschberg's recurrence took place in the orbit six years after enucleation. There can be little doubt that the cause of the recurrence is the presence at the time of the operation of one or more nodules in the orbit, which may be so small as to be quite invisible. The following table, taken from Fuchs' work on sarcoma, shows well the influence which the stage at

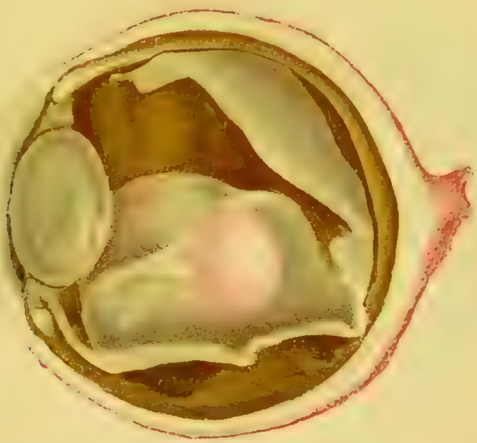

FIG. 101.—Leuco-sarcoma springing from equatorial portion of choroid; appearance immediately after enucleation.

which the operation is performed has on the liability to recurrence:—

Stage.	Total Cases.	Recurrence.	Recurrence Percentage.
I.	21	0	0
II.	97	5	5
III.	117	26	22

It is evident, therefore, as might well be supposed, that the danger is only really great in the third stage. Metastasis is, however, ever so much more common, and constitutes the real source of danger. There are no statistics, so far as I am aware, which give any idea as to the frequency of metastasis; but the proportion of cases where complete recovery has followed the operation, and where the patient has been known to be alive five years afterwards, is not very great. Only one case in thirteen occurred, for instance, in Hirschberg's practice, while another was well two and a half years afterwards. Of ten cases followed by Nettleship, three may be said to have recovered, and one had no return after two and a half years. Fuchs only found recovery in six per cent. of the cases the histories of which he collected. There can be little doubt, however, that the percentage of favourable cases is higher than this.

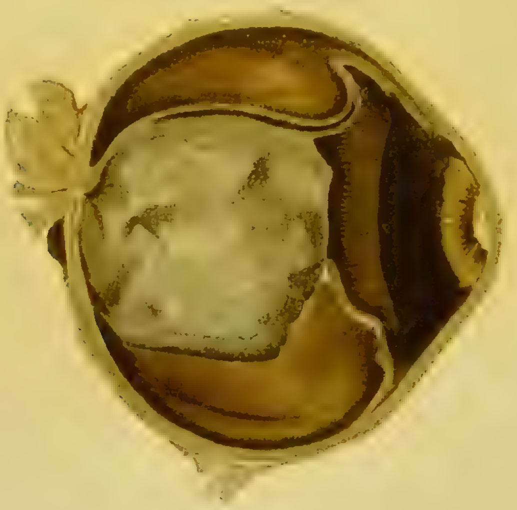

Fig. 102.—Sarcoma of the choroid springing from its posterior portion, showing a slight perforation in the vicinity of the optic nerve. (From a gelatine preparation by Dr. A. Hill Griffith.)

One curious and important point brought out by Fuchs' statistics is that metastasis appears just as likely to take place if the operation be performed at the first as if performed in the third stage, while, as we have seen, it is different with respect to local recurrence.

Later statistics from Leber's practice derived from a total of 25 cases observed from 1874-1889 are in respect to recovery more in accordance with Nettleship's experience, the proportion being 6 to 16. Leber further found metastasis less likely to occur if the primary growth were removed in the first stage. On the other hand he met with two cases of local recurrence, following in one case 3 and in the other 10 years after early enucleation.

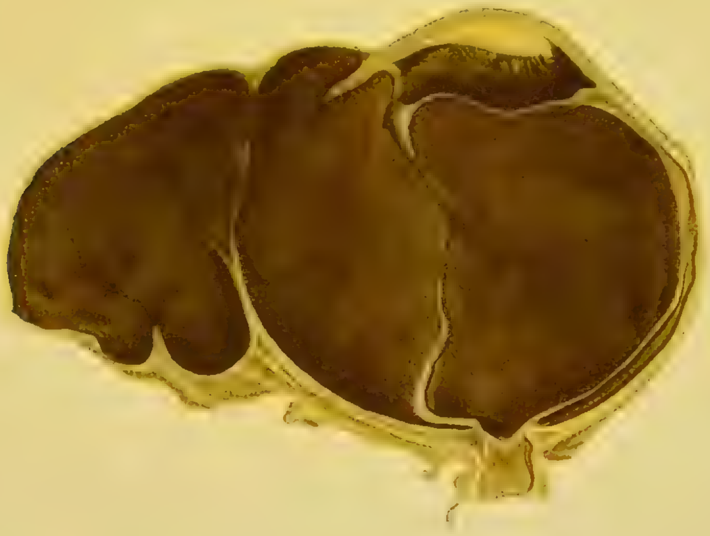

Fig. 103.—Melanotic sarcoma of the choroid in the third stage, the tumour having filled the eye and perforated the sclera. (From a gelatine preparation by Dr. A. Hill Griffith.)

The only possible *treatment* is to remove the eye. This should be done as soon as the diagnosis is certain, in all cases where it is possible to remove the whole growth. In the second stage it is a good precaution to clear out most of the contents of the orbit, as well as removing the eye. This is absolutely necessary where the tumour has extended to the orbit, or is in the third stage of the disease, whereas it would appear from what has been said that enucleation alone is all that is required in the first stage.

It is a disputed point whether or not sarcoma occurs in the choroid as a metastasis. At all events, if it does, it must be so excessively rare as to be of no importance practically.

Metastatic carcinoma of the choroid has been described by several authors—Hirschberg, Perls, Schöler, Manz, &c. It seems always to occur in both eyes, and in the cases recorded has been usually after carcinoma of the mamma.

GLIOMA OF THE RETINA.—Glioma is a white or pinkish-white soft tumour, histologically composed for the most part of round cells, and resembling closely in its structure a round-celled sarcoma,—so much so, that they are by some considered to be identical. On this account it is also called glio-sarcoma, or a sarcoma of the neuroglia. In many respects, however, the clinical features of glioma differ from those of sarcoma when met with in the eye, so that it is well to consider them altogether as distinct diseases. Glioma shows even a more marked tendency to undergo degenerative changes than sarcoma. These may be fatty, calcareous, cheesy, and pigmentary, the first being the most common. As the mass grows it causes a disappearance of the vitreous, and often a loss of transparency of the lens, which, along with the iris, is pressed forwards, causing a more or less marked shallowing of the anterior chamber.

After existing for some time in the retina alone, the choroid and optic nerve become involved in the tumour growth. The growth at first occupies mainly the medullary portion of the nerve, and only after involving this portion for some time spreads to its sheath. This causes in many cases an immense thickening of the nerve, and it is for the most part in this way that extension first takes place to the brain. It is, as a rule, the extension to the brain that causes the death of the patient. Metastasis does occur, but far from being an invariable occurrence, as in sarcoma of the uveal tract, it is comparatively rare. Metastatic glioma has been mostly found in the liver, but has also been met with in the bones, ovaries, kidneys, and lungs.

Instead, then, of four stages which can be distinguished in the clinical history of sarcoma, only three characterise glioma:—(1.) The quiescent or non-irritative stage: (2.) The irritative or glaucomatous stage: and (3.) The stage of extraocular growth.

Blindness is, of course, a very early symptom of the *first* *stage.* It is only comparatively rarely, however, that this symptom directs attention to the disease, as such a large proportion of cases occur in infants, who do not complain until they begin to suffer pain. Often before this the parents observe a peculiar whitish reflection from the eye, not unlike that from the eye of some animals, and what used to be called the *amaurotic cat's eye,* one of the chief causes of which is glioma. This appearance and the accompanying dilatation of the pupil lead to the detection of the disease, and it is then, often long after the first onset of the disease, that advice is sought. .

Owing greatly to the yielding of the sclera, glaucomatous symptoms, or those of the *second stage,* are delayed until a time when the tumour is much larger than a sarcoma, when it passes from the quiescent to the irritative stage. Children are therefore often first brought to the surgeon, when there is already a good deal of increase in the size of the globe, and, what is worse, when the nerve has to a great extent been involved in the tumour growth.

The *third stage,* or the extraocular growth, is ushered in by the perforation usually of the cornea,—sometimes, though much more rarely, of the sclera. Perforation of the cornea is often preceded by a kind of neuro-paralytic and anæsthetic ulceration. As soon as the tumour becomes extraocular it grows with great rapidity, involving the tissues of the orbit, of the temporal fossa, of the cheek, and even of surrounding tissues. The lymph glands in the neighbourhood are sometimes affected, but more frequently not. There can be little doubt that when metastasis does take place, it is brought about by absorption of particles of the tumour into the blood, and not by transmission through the lymph channels.

Occasionally it happens, just as with sarcoma, that an iridocyclitis is set up, and this may lead to shrinking of the eye. Whether or not shrinking takes place, the supervention of an inflammatory attack of this nature introduces a difficulty in the diagnosis.

That form of purulent choroiditis which is met with along with cerebro-spinal meningitis, is the disease most likely to be confounded with glioma. The differential diagnosis will depend partly on the history, and partly on the local appearances. In the case of choroiditis there will usually be a distinct history of an illness; fever, drowsiness, and other more or less marked cerebral symptoms preceding the inflammation in the eye: and when the patient is old enough to observe, the blindness will be said to have come on after the inflammation, and not to have existed before it. The whitish reflex from the fundus very closely resembles that caused by glioma, especially if the lens has lost a good deal of its transparency. When it is possible to make a closer examination of it, the colour is found to be more of a faint straw, and not the pinkish-white or pure white characteristic of glioma. There is, besides, not the marked and irregular convex surface of the mass, or the appearance of retinal and other vessels on its surface met with in glioma. The tension is almost always diminished, and there are also evident signs of inflammation—synechiæ, uveal pigment on the lens capsule, &c. An important point to observe, too, is the condition of the periphery of the iris. When there are purulent deposits in the vitreous, the contraction which takes place in them leads usually to retraction of the peripheral portion of the iris, and deepening of the anterior chamber in this position. In glioma the tension is rarely low, and may be distinctly increased even before glaucomatous symptoms become evident. The shallowing of the anterior chamber takes place throughout, and is due to the pushing forwards of both lens and iris. The differential diagnosis between glioma and sarcoma has already been discussed.

Glioma is less frequent than sarcoma,—according to Horner's statistics, about twice as uncommon. Twenty-three cases occurred amongst 75,000 persons, or one in every 3260. Of 252,347 patients attending during five years at Moorfields and Dublin, and during four years at the Manchester and Birmingham Eye Hospitals, 95 had glioma—a proportion of one to 2660. It is much more common in infants, but has been met with up to the age of twelve. Possibly it may sometimes occur at a later age.

The few cases which have been described as occurring in adults have been either undoubtedly or very probably cases of sarcoma involving the retina. Cases have occurred where the tumour has been intra-uterine. Hirschberg even believes that the first beginnings as it were of the tumour are always congenital, and that the growth starts in earnest at different times after birth. There is certainly nothing very improbable in this view. Of twenty-five cases treated by Horner three were congenital, six occurred in the first year, five in the second, three in the third, two in the fourth, and one each in the fifth, seventh, and eighth years respectively.

The children affected are otherwise healthy, and so are their parents, as a rule; there does not appear to be any heredity. There is, however, a very marked tendency to several members of the same family becoming affected with the disease, though it is rare that this predisposition extends to all the members of a family. Occasionally both eyes are affected, and generally then both at the same time, or one shortly after the other. On this account alone it is evident that the disease in the second eye is independent of the first, and does not spring from a continuity of the growth along the chiasma But there is absolute proof of this independence in the fact that complete recovery may take place after removal of both eyes for glioma.

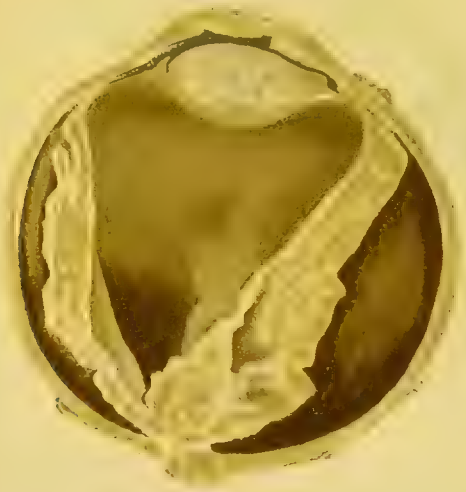

Fig. 104.—Case of glioma of the retina. (From a gelatine preparation by Dr. A. Hill Griffith.)

The *prognosis* in glioma is always bad, and altogether hopeless if the disease be left to its own course. It was generally thought, too, before von Graefe's time, that it was incurable. This was no doubt because the operation was undertaken too late—in the third stage of the disease—and probably often very imperfectly performed. With improved methods of examination, and by operating as soon as the diagnosis was certain, Graefe was soon able to record cases in which complete recovery resulted from operation. Although the disease is extremely malignant, it is mainly so, as has already been said, on account of the great tendency to extension to the brain. If an opportunity occurs of operating at an early stage, there is a fair chance of eradicating it. It is always advisable to remove as long a portion of the optic nerve as possible when there is much thickening of it. If the operation be done during the glaucomatous or later stage, the whole contents of the orbit should be removed. Even with this precaution the chance of recurrence is very great,—quite as great as in the case of sarcoma, if not more so. The prognosis is therefore considerably more favourable than in sarcoma if the operation be performed early, while it is, if anything, worse in the third stage.

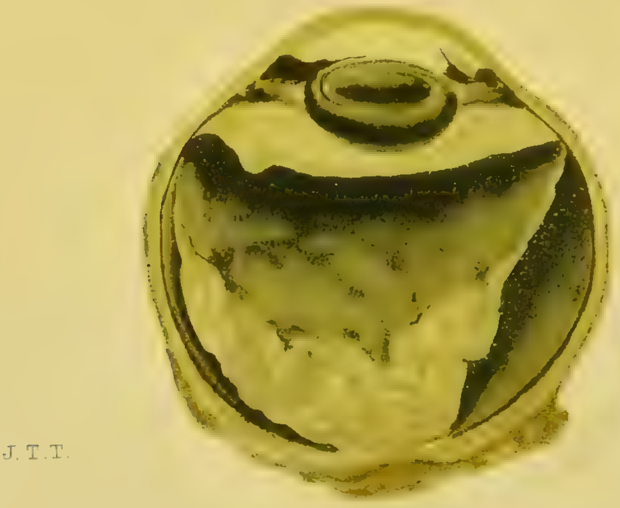

Fig. 105.—Glioma of the retina complicated with pseudo-glioma. (From a gelatine preparation by Dr. A. Hill Griffith.)

I know of two cases in which there has been complete recovery, but as I have not kept notes of all the cases I have seen, I cannot say what proportion they constitute, nor would the proportion given by such a small number be of much value. Almost all ophthalmic surgeons to whom I have spoken on the subject, however, have had cases of recovery, and as the number of cases treated by any one surgeon could at the best not be very numerous, as the disease is considerably less frequent than sarcoma, this of itself shows that a cure is by no means a very rare event. Of four cases recorded by Nettleship, one recovered certainly, and one other probably. Of eleven cases followed by da Gama Pinto, two recovered. Of Horner's twenty-three cases, I gather from Vetsch's account that sixteen died, and only four were known to recover. Other cases of recovery have been recorded, notably one by Agnew after enucleation of both eyes.

Glioma of the retina is a disease which has long been known at a late stage of its development. Wardrop, who was the first to show that the large growths, to which the disease gives rise when not interfered with, spring from the retina, called it *fungus haematodes*. He was also aware of its malignant nature, and of its spreading along and involving the medullary portion of the nerve. In France it has for long been known by the name of *encephaloid* of the retina, given to it by Lænnec. Virchow was the first to show that the point of origin is the connective tissue or neuroglia of the retina, and hence the name glioma. Our present knowledge on the subject is greatly owing to the clinical investigations of von Graefe and Hirschberg. Different layers of the retina are first implicated, and partly owing to this, partly also to the manner of extension, the main growth either occupies the vitreous chamber without leading to detachment of the retina, or it causes detachment, and occupies the space between the retina and choroid. The first form is less common, and is known as *glioma endophytum*. The second and more common form often receives the name of *glioma exophytum*. Of seventy-seven cases collected by Hirschberg, only two were cases of glioma endophytum; but this form occurred in four of eleven cases recorded by da Gama Pinto from Heidelberg. Hirschberg's proportion probably, therefore, hardly represents the actual one.

CHAPTER XIII.

DISEASES OF THE ORBIT.

MOST affections of the orbit have one characteristic symptom in common. They give rise to more or less protrusion of the eyeball,—*proptosis* or *exophthalmos.* An abnormal sinking of the eye back into the orbit, or *enophthalmos,* is rare, apart from that which occurs in very old people owing to the disappearance of the orbital fat.

Diseases of the orbit are, on the whole, of rare occurrence. From the statistics published by the Royal London Ophthalmic Hospital, the Manchester and Birmingham Eye Hospitals, and the National Eye and Ear Infirmary, Dublin, I find that amongst over 250,000 patients who presented themselves for treatment at these institutions during the space of four and five years, about one in 800 was the subject of some orbital affection.

The causes of exophthalmos are—(1.) Inflammation of the orbit; (2.) Tumour of the orbit; (3.) Aneurism of the orbit; (4.) Graves' disease; and (5.) Injuries to, and foreign bodies in, the orbit.

INFLAMMATION OF THE ORBIT.—Although it is not always possible to make the differential diagnosis, it is important to make a clinical distinction between an inflammation beginning in the periosteum of some portion of the orbital wall or *orbital periostitis,* and one which originates in, or is confined to, the connective tissue of the orbit,—*orbital cellulitis.* While the former sooner or later extends to the cellular tissue, an original cellulitis does not lead to inflammation of the periosteum or bone.

Cellulitis is the more common affection, being set up, as a rule, by some trauma or by the extension of a panophthalmitis, originating often in some severe corneal inflammation. Of purely idiopathic inflammations, however, it should be remembered that periostitis is much more frequent than cellulitis. The periostitis may be an extension from some other part, and may, for instance, be set up by a decayed tooth, or it may originate in the orbit, in which case there is usually some strumous or syphilitic cause for its appearance. The periostitis may be merely hyperplastic, or it may be gummatous, and lead to the destruction of the tissue involved.

The surrounding cellular tissue and fat frequently participate in the inflammation which has originated in the periosteum, and a more or less localised abscess results, which gives rise to proptosis and great pain. When the case is originally one of periostitis, and the protrusion of the eyeball great, then it may be looked upon as certain that it has proceeded to suppuration. Sometimes fluctuation can be felt in the orbit, but this is not always the case. The pain is very great, and subject to extremely acute exacerbations, which are apt to come on at night. In many cases pressure over some part of the bone surrounding the orbit gives rise to pain, especially if the pressure be made with the finger passed as far to the inner surface of the orbital margin as possible and directed away from the eye. In this way a periostitis may often be distinguished from a cellulitis. If, however, the site of the inflammation be very far back, there may be little or no pain on pressure over the bone.

In exophthalmos due to periostitis, the protrusion generally takes place more rapidly than when the cellular tissue alone is inflamed, while the restriction of movement which accompanies the inflammatory protrusion is often less absolute in some directions than in others, and the displacement itself not directly forwards, but more in some particular direction, often downwards or inwards. In orbital cellulitis, on the other hand, the pain is not so great as a rule, a considerable degree of protrusion often taking place with little, or only what is described as a dull pain being experienced; the proptosis takes place more gradually, and is generally directly forwards without any greater deviation in one direction than another, while the restriction in the power of movement is equally marked in all directions, and is often practically absolute. There is no pain on pressure over the bone, but pushing the eye backwards gives rise to more or less pain. In both periostitis and cellulitis there is more or less chemosis, as well as swelling and redness of the lid. These external indications of inflammation are said by some to be greater in periostitis, but I do not think that the prominence of the symptoms can be relied on as affording information of much diagnostic value. The state of vision in the protruding eye is a point of more importance, as in cases of idiopathic cellulitis, in which alone, as a rule, there can be a doubt as to the diagnosis, it is more frequently seriously affected or altogether destroyed than in periostitis.

In some cases of gumma the swelling is so hard that it might readily be taken for a tumour. These occur most frequently on the roof of the orbit, not very far from its margin. Periostitis, as well as caries, is also met with confined to the margin of the orbit, or at all events not spreading far back. This, like the more serious deep-seated inflammation, is mostly strumous, rheumatic, or syphilitic. The most common sites for it are the lower ridge and external angle of the margin of the orbit. It causes great œdema of the lids and chemosis, the conjunctiva being infiltrated with a clear, translucent, straw-coloured fluid. The site of periostitis is easily discovered by pressing with the finger over different parts of the orbital margin. This form less frequently, in my experience, goes on to suppuration than the deeper affection. I have met with it on several occasions symmetrically affecting both sides.

When an abscess does form it should be opened antiseptically. In the deeper-seated abscesses, too, it is important not to delay too long in making a free opening for the escape of the pus and free drainage. Besides, by direct transmission through the diseased bone forming part of the orbital wall, cases which have been left to themselves have been known to cause death by bursting through the back of the orbit. Other positions in which a spontaneous evacuation of the pus may take place are—externally through the lid and conjunctiva, which fortunately seems to be most common, or into the cavity of the nose or the antrum, but this probably only where the intervening bone is involved.

The caries or necrosis, so often associated with the periostitis, causes the healing process to be usually a protracted one, so that a fistulous opening may remain for months. Where such a process takes place at the orbital margin, the eventual cicatrisation is apt to lead to ectropion. Children are especially liable to periostitis of the orbit, both in the superficial and deep parts.

Often it is apparently set up by a trauma, though probably there is generally a strumous or inherited specific taint which favours its occurrence.

The frequent connection between severe inflammatory conditions of the coats of the eye and the cellular tissue of the orbit has already been referred to. It is the surrounding connective tissue or Tenon's capsule which first participates in the inflammation, but there seems no reason to suppose that it is usually confined to that part, and consequently to distinguish, as is done by some surgeons, between tenonitis and more general orbital cellulitis.

Idiopathic orbital cellulitis is due either to thrombosis of the cavernous sinus or to an inflammation which is set up by some specific poison, which, somehow or other, finds its way to the tissues of the orbit.

The inflammation thus originating in the orbit may extend to the brain. When this takes place it is probably always, as was first pointed out by Berlin and confirmed by Leber, through the vessels, and not through the lymphatic channels. However it may originate, an idiopathic inflammation of the orbital tissues is much more liable to cause either complete destruction of, or grave interference with, vision, than that which results from accident, or from the extension of a purulent choroiditis. It is unlikely, therefore, that the mere compression of the nerve, which might be supposed to take place when the eyeball is greatly protruded, is the main cause of this disastrous complication, but that the nature of the inflammation itself seems of greater importance in this respect.

The danger to life is also greater in the idiopathic cases. Indeed, those which primarily depend on thrombosis of the venous sinus are always fatal, and usually the exophthalmos is at the same time double. How it is that in some cases which have a peripheral origin, a spreading of the inflammation to the brain, and consequently generally a fatal termination, occurs, while in other apparently similar cases this complication is not met with, does not seem quite clear.

The affection often begins with fever, and more or less marked indications of gastric irritation; not infrequently there is an evident erysipelas of the face and head which has spread to the orbit, or a lesion of some kind in the neighbourhood of the eye, which has given rise to phlebitis.

Probably in both cases the result is a more or less extensive thrombosis of the veins in the orbit, which may extend to the central vein of the retina, and cause blindness, or, by extending backwards, lead to phlebitis and thrombosis of the cavernous sinus. Often, however, no such tangible cause can be discovered. In such, Leber assumes, nevertheless, the existence of some similar infectious cause. He says "that the origin of the orbital inflammation is to be regarded in the same light as probably every one now looks upon the occurrence of an apparently spontaneous attack of erysipelas, viz., that infectious matter finds its way into an unnoticed abrasion of the skin. The wound thus infected is probably generally in the lid, which, owing to its abundant venous supply, offers particularly favourable conditions for the establishment of an infected thrombus." While an orbital cellulitis of this nature is usually confined to one eye, it has occasionally been met with on both sides. The cause of the bilateral occurrence has been found in the case of the erysipelatous variety to be either an extension of the thrombosis into the cavernous sinus, and from thence into the other orbit, or—and this seems to have been more frequently observed—the inflammation of the skin may spread into both orbits.

In orbital cellulitis caused by erysipelas, death occurs from extension to the brain in probably about twenty-five per cent., and is more likely to occur when both orbits are affected than where the exophthalmos is on the one side alone. It is considerably more likely than not to be followed by complete blindness. In the cases I have seen, the subsequent atrophy of the optic disc has been extreme, and the vessels much diminished in number and calibre, but I have not observed any marked degree of vasculitis or perivasculitis leading to dense white lines in the place of the retinal vessels, such as have been described by Knapp and others.

The *treatment* of inflammatory exophthalmos must be directed as far as possible to preventing the retention of pus in the orbit. Before the protrusion of the eye is great, and so long as actual suppuration has not taken place, the treatment should be mainly expectant, or at all events no active local treatment is required. Constitutional remedies suited to the case may be tried, and a firm bandage applied over the eye. It should be remembered that some cases disperse without leading to abscess. This favourable result is not to be expected when the pain is excessive and the exophthalmos marked, and the formation of the pus should then rather be favoured by frequent antiseptic poulticing. As soon as any fluctuation can be made out, an incision should be made into the orbit, the site for the incision being chosen according to the position of the most evident fluctuation, if such exist, or where the separation of the eye from the orbital wall is most pronounced. The knife—either a large Graefe's knife or a narrow Sichel—should be plunged deeply into the orbit, with the edge turned away from the eye. The escape of pus causes great relief to the patient, and generally very considerable, though not complete, reduction of the proptosis. It is necessary to secure efficient drainage afterwards, as in most cases, and especially where there has been periostitis, the discharge will continue for a long time. Careful probing may be made through the wound from time to time in order to determine the state of the bone, especial care being taken where any diseased bone is found in the roof of the orbit.

EMPYEMA OF THE FRONTAL SINUS.—A collection of matter may take place in the frontal sinus on one or both sides. This is by no means common, the bilateral affection being especially rare. The symptoms to which an empyema in this situation gives rise depend upon its acuteness and the degree of patency of the normal opening which connects the sinus with the nose. Most cases have probably lasted a long time before they lead to any swelling, other than possibly some degree of distension of the cavity, which remains unnoticeable. After a longer or shorter time the anterior bony wall becomes perforated at its orbital aspect, where it is thinned, by the retained secretion, and a rounded elastic swelling makes its appearance at the upper and inner angle of the orbit, causing epiphora, as it increases in size, from pressure on the tear sac, and also some degree generally of displacement of the eye, with a corresponding diplopia in certain directions of fixation. The displacement of the eye is forwards, downwards, and outwards. Occasionally, however, the diplopia is more marked than can be accounted for by this displacement, which may be very slight, and is then no doubt partly or wholly due to interference with the action of the tendon of the superior oblique muscle. Sometimes the patient has for long complained of headache, confined to, or starting from, the region of the sinus; at other times the condition is found to have given rise to very little discomfort. Notwithstanding the bursting of the abscess through the bone, the passage to the nose may frequently be found to be not altogether obliterated, as by pressure over the swelling the contents may often be emptied into the nose.

Occasionally, as in a case recorded by Knapp, bursting takes place backwards, leading, as it does in abscess in the mastoid cells, to a cerebral abscess. This, however, is fortunately a very rare occurrence.

Frontal sinus abscess may occur at any time of life, except in young children, as the sinuses are not developed until after the fifth or sixth year. A distension of the sinus may also take place, owing to the extension of nasal growths into it. Tumours do not, however, appear to grow primarily in this situation. Only one case has been recorded by Knapp where distinct masses of a myxomatous nature, unconnected with any similar development in the nasal passages, were found complicating the condition of abscess which called for surgical interference.

The cause of distension of the frontal sinus is not always very clear. Sometimes there has been many years previously an accident which has possibly closed up the passage to the middle meatus of the nose, but as a rule such an interference with the outflow of the secretion from the sinus seems more probably to be due to swelling of the mucous membrane lining the channel. In the first case the pent-up fluid slowly increases; in the other there is at the same time an inflammatory condition of the lining membrane leading to suppuration in much the same manner as that commonly met with in the tear sac.

The *treatment* consists in cutting down on the swelling, enlarging if necessary the aperture in the bone, and then keeping up a proper drainage by inserting a drainage tube and syringing out the cavity at first twice daily, and afterwards less frequently. In cases where the discharge is very purulent at first, there is generally no actual stricture of the passage to the nose, and nothing further is required.

When there is no passage one may be made with a small gouge, and kept open by frequently passing a probe through it, or the following operation described by Lawson may be performed. With the index finger of one hand in the sinus, the right in the case of the left and the left in the case of the right sinus, the little finger of the other hand is passed up the nostril, "and an endeavour made to find out the spot at which the tip of the finger in the sinus will approximate most closely to the one in the nose. After a little search it will be found that at one part the fingers will almost meet, there being only a thin plate of bone between them. Having gained this information, the finger in the frontal sinus is to be withdrawn, but that in the nostril to be retained *in situ*, to act as a guide to the gouge or elevator which is to be passed into the sinus, and made to force a passage into the nose through the lamina of bone on which the tip of the little finger is resting. A communication between the frontal sinus and the nose having been thus established, an indiarubber drainage tube, with holes cut at short distances, is to be introduced, one extremity of which is to be afterwards fastened on the forehead, whilst the other end protrudes slightly from the nostril. The easiest way of introducing a drainage tube is to pass a probe with an eye up the nostril and out of the wound, and having fastened the tube to it by means of a piece of string, to draw it back again through the nose."

As a rule, it is long, sometimes a year or more, before the discharge stops altogether, and before it is safe to allow the opening to close. Sometimes a permanent fistula is left. Leber has described a visible pulsation in the fluid contained in the sinus in a case on which he operated. Such pulsation, which might possibly give rise to the idea of a communicated pulsation from the cerebral vessels, is really, as Boeckel has shown, always met with where there is a narrow opening into a rigid cavity lined by a vascular membrane.

Suppuration in the ethmoidal cells, originating in some nasal inflammation, may give rise to abscess of the orbit. In such cases pus is discharged through the nose, and the fluid injected into the wound made in the orbit after the abscess has been opened escapes in part by the same channel. Suppuration beginning in this situation is the probable cause of such cases of orbital cellulitis as appear to be connected with affections of the nose. We have seen, on the other hand, that an abscess originating in the orbit may, if not opened, burst through the ethmoid into the nose. Priestley Smith, Eales, and some older writers have recorded cases of caries and necrosis of the ethmoid leading to exophthalmos. Cases have been described, too, and are referred to by Berger in his *Diseases of the Ethmoid and Sphenoid*, in which exophthalmos has been a symptom of similar diseases of the sphenoid. In these cases the optic nerve may be pressed on or inflamed, and thus an extension of the disease to the optic foramen may be the cause of blindness. Finally, as a curiosity, may be mentioned a case described by Mair, in which an empyema of the antrum spread first to the ethmoid cells, and then to the orbit, causing death by meningitis.

TUMOURS OF THE ORBIT.—The orbit may be the seat of both simple and malignant new growths, and these again may primarily form in the orbital tissues, or invade them by extension from neighbouring parts. Some malignant tumours in this situation are metastatic.

Although, from a clinical point of view, the most satisfactory division of orbital tumours is into simple and malignant, it seems more convenient to consider them according to the structures from which they spring. The tumours which may be recognised according to such a classification are:—(1.) Tumours of the bony wall of the orbit; (2.) vascular tumours of the orbit; (3.) tumours of the connective tissue; and (4.) tumours of the optic nerve. Tumours of the lachrymal gland are of course, properly speaking, also orbital. They have, however, been referred to in the chapter on affections of the lachrymal apparatus.

Orbital tumours are, comparatively speaking, of rare occurrence. Of 2058 tumours met with by Billroth, 217 occurred in the face and neck, and of these only 18 were in the orbit. Tumours originating in any part of the orbit, though they may lead to the destruction of vision—occasionally, when they set up inflammation, to destruction of the eye itself—do not spread to the eyeball. A few exceptions are met with in tumours growing from the optic nerve, but these are extremely rare. When, therefore, a growth is seen to occupy both the globe and the orbit, as is by no means uncommon, it may be inferred that the starting-point has been the eye itself. As they extend within the orbit, new growths lead to more and more exophthalmos, and at the same time to more or less marked interference with the movements of the eye, giving rise consequently to diplopia as long as the vision is sufficiently retained in the displaced eye. The direction of greatest displacement affords an indication, too, of the position occupied by the tumour. Growths of the nerve itself or its sheaths, or growths which lie within the cone of muscular fibres which extends from the apex of the orbit to the eyeball, cause less interference at first with the movements of the eyeball, and at the same time displace it directly forwards. In the case of growths springing from other parts there is often a preponderance of displacement in some particular direction. Great differences are met with in the amount of pain caused by a tumour of the orbit, the malignant growths being on the whole more painful than the benign tumours, but not so markedly so as to render the degree of pain a point of much practical diagnostic importance. The vision of the eye may remain perfectly good, notwithstanding considerable proptosis, but many orbital growths sooner or later lead to its destruction. The vision is generally lost by neuritis or optic atrophy from pressure, rarely, if ever, owing to the nerve being actually involved in the tumour growth.

TUMOURS OF THE BONY WALL OF THE ORBIT.—Osteomata or exostoses of the orbit are exceedingly rare. I find that out of 128,000 cases of eye affections treated at Moorfields Hospital during five years, eight were cases of this nature. Knapp found four cases amongst 56,000 patients. The frequency may therefore be taken at about one in every 15,000 patients seeking advice at an ophthalmic hospital or ophthalmic department of a general hospital.

The tumours consist of excessively hard close bone, like ivory exostoses elsewhere. They are mostly found at the upper and inner angle of the orbit growing from the frontal bone, while next in point of frequency are those growing from the ethmoid. But they may apparently occur at any part of the orbit. They usually grow very slowly, so that years may elapse after they have first begun before they attain any size. The extension either takes place into the orbit alone, causing a gradual displacement of the eye, to which the eye may accommodate itself to a great extent, but which eventually causes its destruction, or it may take place at the same time into the cranial cavity or the maxillary antrum, according to the situation of the growth.

The *etiology* of exostosis seems to be altogether obscure. There is generally not much difficulty about the *diagnosis*—the slow growth, excessive hardness, immobility, and direct connection with the bone being sufficiently characteristic. I have, however, once seen a mistake made in the case of bone thrown out round a chronic abscess in the ethmoid. If left alone they do not appear to destroy life, even if they at the same time grow into the cranial cavity. Their gradual enlargement may, however, lead to eventual destruction of the eye.

Treatment.—Frontal exostoses may be left alone as long as they do not interfere with vision by pressure on the eye. When this takes place they can be removed with almost complete safety by drilling a number of holes through their base, where they are usually thinnest, with a dentist's drill, and completing the separation by means of a small circular saw attached to the same apparatus, and a hammer and chisel. The latter should be used with as little force as possible, as it may lead to fracture of the bone from which they grow, unless the neck of the tumour has been sufficiently weakened by the perforations. By this means the exostoses are not completely removed, and a repetition of the operation may be necessary after the lapse of some years. Complete extirpation has often been effected, but is risky in all cases where there is a possibility of some of the tumour extending into the cranial cavity. It has often—according to Berlin in twenty-five per cent. of the recorded cases of exostosis of the frontal bone—been followed by a fatal result from injury to the brain. The results of extirpation of exostoses of the ethmoid have been much less unfavourable.

VASCULAR TUMOURS OF THE ORBIT.—Nævi are occasionally met with extending from the skin of the face into the orbit.

Some years ago I removed at the Edinburgh Infirmary, with the assistance of Dr. MacGillivray, a tumour entirely composed of blood-vessels, which extended from behind the globe forwards under the conjunctiva, thus embracing the complete inner half of the eye, which it nearly equalled in volume. It was not connected with any vascular dilatation on the skin, and apparently received its blood supply from the angular artery. It had been growing for many years. There has been no recurrence during the six years which have elapsed since the operation, and the eye has perfect vision, though it is considerably restricted in its movements inwards from the cicatricial tissue which has formed in the position of the anterior portion of the growth. I have seen one other very similar case, where, however, the tumour was not so large, which came under the care of Dr. Duncan at the Edinburgh Royal Infirmary, and was treated by him by electrolysis.

Cavernous vascular tumours have been described, but are very rare. They appear to be most commonly situated within the cone of ocular muscles. They have all been of excessively slow growth, and are characterised by more or less marked differences in the degree of exophthalmos to which they give rise, according to the extent to which the erectile tissue of which they are composed is engorged. The existence of such a tumour might be suspected, if, by any condition which gave rise to engorgement of the vessels of the head, the exophthalmos was distinctly increased in amount. They can, too, be generally emptied by pressure on the eye. They are painless and of exceedingly slow growth, if not sometimes altogether stationary. Occasionally, by the bursting of a vessel and the consequent extravasation of blood, a sudden increase in the exophthalmos may take place from the formation of a sanguineous cyst, as in a case recorded by Lawson. Phleboliths have also in some cases been found embedded in the tumour.

Förster and Wiesner have described cases which appear to have been lymphangiomata. In these there was an absence of compressibility and of temporary engorgement, but interference with the movements of the eyeball.

The best *treatment* for such tumours, as well as for nævi in the orbit, is probably electrolysis.

TUMOURS OF THE CONNECTIVE TISSUE, &C. OF THE ORBIT.—These are simple or malignant, the latter being more common.

The simple tumours met with are cysts and lymphadenomata. The cysts are either dermoid or hydatid.

I have not met with a case of dermoid cyst of the orbit. One case was operated on by Walker in the Edinburgh Royal Infirmary in 1867. They appear, however, not to be extremely rare: Berlin, from whom the following account is taken, collected records of seventy-three cases, and a few have been described since his monograph was published. They are fœtal structures, with contents similar to those met with in the case of similar cysts elsewhere, only with a tendency to greater fluidity. While some have contained hairs, epidermic scales, and fatty matter, and even in one instance a tooth, others have been filled with a more fluid albuminous material. They are usually unilocular, but sometimes divided into cavities, and have been met with of all sizes up to that of a goose's egg. They lie outside the muscular cone, and are sometimes pretty firmly attached to the apex of the orbit. They slowly increase in size, generally being large enough to attract attention before the patient reaches the age of twenty.

The *diagnosis* is difficult in all cases, but least in cases where there is fluctuation, and a history of the condition being probably congenital. With the exception, perhaps, of some true optic nerve tumours, the congenital conditions which cause exophthalmos are only dermoid cysts and angiomata. As they go on increasing in size, the *treatment* consists in removing them as thoroughly as possible.

A curious variety of orbital cysts has been met with by Talko and others in cases of anophthalmos and microphthalmos, which appears to be a cystic degeneration of more or less of the embryonic structures which should go to form the eye. This is, however, only a curiosity and of no practical importance, besides being of very rare occurrence.

Hydatid cysts are comparatively rarely met with in the orbit, and in this country, where the echinococcus is altogether not very frequent, exophthalmos due to this cause is probably much rarer than that resulting from a dermoid cyst. The diagnosis cannot well be made with any certainty; probably, in most cases fluctuation can be made out, and the fact of the exophthalmos being only recent might help to differentiate it from a dermoid cyst. An exploratory puncture should be made in a doubtful case.

The only case which I have had the opportunity of seeing occurred at Moorfields, and was there operated on by Lyell. In that case neuritis and optic atrophy preceded the exophthalmos, which eventually became so great that sloughing of the cornea began to take place, when the patient, after an absence of some months, presented himself again at the hospital. Evisceration of the orbit was then decided on, and the eye first removed. On preparing to dissect out the tumour with scissors the wall of the cyst was opened, and the fluid gushed out, and was followed by complete collapse of the growth. These cysts have been met with in all parts of the orbit. They generally give rise to pain, and appear to be more common before than after twenty. Perforation of the cyst into the cranial cavity has been described.

Only a very few cases of cysticercus in the orbit have been described, two of which have occurred in this country, and were recorded respectively by Bowman and Higgens.

LYMPHADENOMATA.—A few cases of non-malignant primary symmetrical tumours in both orbits, the site of which appears to have been outside the cone of muscles, and near the periosteum, have been described by Leber, Gayet, and others. The blood in some of these cases has been found to contain a great excess of white corpuscles, their proportion to the red being as one to five or one to four. They have been described, and no doubt correctly, as lymphadenomata. The ages of the patients in the cases so far recorded have ranged from four to seventy. The prognosis is bad. In one case the tumours disappeared spontaneously previous to the death of the patient: in two others a cure was effected by extirpation, which there is good reason, however, to believe might not always be possible with the retention of the eyes, and therefore hardly justifiable except when they may have already produced blindness. The disease is one which, though rare, deserves further attention.

MALIGNANT TUMOURS.—The primary malignant tumours of the orbit are mainly, if not entirely, sarcomata. Carcinomata do occur, but, except in the cases of tumours of the lachrymal gland, they are probably always metastatic. They seem, too, to be of rare occurrence; only one case after scirrhus of the mamma has come under my observation, and was shown to me by Dr. Cotterill of Edinburgh. Primary sarcoma of the orbit is usually non-pigmented, but melanotic sarcoma has been met with originally growing from the orbital fat. It is most frequently, however, an extension from an intraocular tumour. Various forms of sarcoma occur in the orbit, much the most frequent being the round and spindle-shaped varieties. The rapidity of their growth depends partly upon the tumour itself, and partly on the age of the patient. Round-celled sarcomata, as elsewhere, are the most malignant, while the younger the patient the greater is the tendency to rapid growth. The tumour may extend to the brain, and also to the antrum, temporal fossa, &c. Sarcoma of the orbit is usually painful, more so, at all events, than most simple orbital tumours. It is usually fatal, although sometimes early removal may altogether eradicate the disease. Occasionally the growth stops spontaneously, and the tumour undergoes various degenerative changes. This is, however, an occurrence of such rarity that it can hardly be regarded as of any practical importance. The *treatment* consists in removing the contents of the orbit as thoroughly as possible.

TUMOURS OF THE OPTIC NERVE are of rather rare occurrence. They have been divided by Leber into true or *essential* and *non-essential* optic nerve tumours. The first are those which spring originally from the nerve itself; the second spring from the sheath, involving the nerve secondarily. Only the first are here referred to. Altogether, probably not more than seventy cases have been recorded. During the last ten years three cases have been met with at the Edinburgh Royal Infirmary. One tumour was removed by Dr. Argyll Robertson; the other two happened to be under my care. Tumours of the optic nerve have mostly occurred in early life, being possibly sometimes congenital. As the muscles are not involved in the tumour, the movements of the eye are generally relatively good, even when the proptosis is considerable. The eye, though as a rule soon rendered sightless, remains intact until the protrusion is so great that it cannot be covered with the lids. Until the cornea becomes affected from exposure, there is complete absence of pain. A very considerable degree of antero-posterior flattening of the eye may take place as the tumour increases in size. The eye, when enucleated, may thus have an appearance which is different in this respect from that caused by other orbital tumours. Curiously enough, no subjective sensations of light have been recorded, although the neuritis and atrophy which accompany the growth of the tumour show that the nerve fibres must be pressed upon.

A suspicion that a tumour in the orbit was of this nature should be raised by the movements of the eye being good, by the early though not absolutely sudden loss of vision, and by the comparative absence of pain; but the *diagnosis* cannot always be made with any certainty. In some cases of sarcoma of the orbit, sudden blindness may come on, probably owing, as in a case of Leber's, to compression from hæmorrhage.

The progress of these cases of optic nerve tumour is very slow, generally lasting for many years. Most have been myxomata, or combinations of other forms of tumour with mucoid tissue. Next in frequency come fibromata, as in a case recorded by Brailey. A true neuroma has only once been described by Perls. The tumours have as a rule not involved the outer sheath of the nerve. The result of operation has not generally been satisfactory. In one of the cases in which I removed, and which has been recorded by Sym, no recurrence took place. The tumours are always found to be encapsuled; but, unless removed at a comparatively early stage, may have spread too far backwards along the nerve to admit of complete removal.

TUMOURS WHICH EXTEND TO THE ORBIT FROM ADJACENT PARTS.

ENCEPHALOCELE.—Of ninety-three cases of encephalocele, the records of which have been collected by Haab, sixteen occurred in the naso-frontal region. Some of these did not extend into the orbit, so that encephalocele in that situation is certainly a very rare occurrence, although the possibility of its existence should be borne in mind.

The *diagnosis* could hardly be a matter of difficulty, when, as has mostly been the case, the appearance presented is that of a fluctuating, somewhat transparent, and more or less distinctly pulsating tumour at the inner angle of the orbit, pressure on which causes its disappearance, and at the same time gives rise to symptoms of cerebral irritation. The history, too, of its being congenital is of diagnostic importance. In some cases, however, there have been complications which have rendered the diagnosis difficult; thus the encephalocele has been completely shut off from the cranial cavity so as to form a true cyst, or the skin covering it has been abnormally supplied with blood-vessels, giving rise to the appearance of a nævus. In such cases the appearance of hydrocephalus, as Berlin suggests, would weigh in favour of the growth being an encephalocele, and further evidence would be afforded by the condition being bilateral, or by the presence of an undoubted encephalocele in some other situation. The large and rapidly growing tumours of this nature soon prove fatal, generally in the first few weeks, so that it is only in the case of the smaller ones, where it becomes possible for the patient to attain to full growth, that there is likely to be any doubt as to its nature. In any case it is probably safest to avoid interference.

NASAL POLYPI sometimes extend into the orbit and cause displacement of the eye. I have seen a case of this nature along with Mr. Miller of Edinburgh. More or less complete blindness is thus produced by pressure. Sometimes, as in a case recorded by Priestley Smith, the pressure seems mainly to be exerted at the optic foramen, owing to the erosion of the sphenoid by the myxomatous growths.

Tumours of the ethmoid, sphenoid, and antrum may spread to the orbit. Primary sphenoidal tumours appear to be very rare.

In a case of a woman of fifty, under Dr. Affleck's charge at the Royal Infirmary, in which I had the opportunity of seeing both the symptoms during life and the appearances on *post-mortem* examination, a carcinoma growing from the sphenoid passed into both orbits, causing first neuritis and atrophy, and afterwards proptosis. A mass of the tumour was also found to have passed into the cranial cavity, and yet the only marked symptom which was observed until shortly before death, which took place from meningitis, was polyuria. In other cases there has been a similar absence of cerebral symptoms, but more frequently the patients have been subject to epileptiform attacks.

TUMOURS OF THE LIDS AND SKIN OF THE FACE—epithelioma principally—may spread eventually into the orbit and cause exophthalmos.

In one case, which was for several years under my treatment, a lupus of the face extended to the eye and orbit, forming a mass on the eye as large as the globe itself. I first removed the eye and contents of the orbit, and scraped away with the sharp spoon and knife the lupus of the skin of the cheek and side of the nose. Two years afterwards the patient returned with a recurrence of the growth in the orbit, antrum, and temporal fossa, which I removed with portions of the bone of the face, but not thoroughly, as some months afterwards, having decided, on consultation with Mr. Annandale, that the only chance was excision of the upper jaw, he performed that operation after a preliminary tracheotomy, and very completely cleared away the tumour from all its apparent connections, even the body of the sphenoid. The patient, however, died of pneumonia some days after the operation. In this case, as was suggested by Dr. Jamieson, who saw it, and as was confirmed by microscopic examination, the lupus evidently had undergone carcinomatous change.

ANEURISM OF THE ORBIT.—Two very important monographs on the subject of aneurism of the orbit have appeared in recent years, the first by Rivington, in the fifty-eighth volume of the *Medico-Chirurgical Society's Transactions*, and the second by Sattler in the *Handbook of Ophthalmology*, edited by Graefe and Saemisch. To these, and especially to the latter, I must refer the reader for a discussion of many points of difficulty and uncertainty in connection with the pathology and diagnosis of this condition.

Aneurism of the orbit is certainly a rare affection, though probably not so rare as would appear from the number of cases published up to the time of Sattler's monograph. Sattler collected records of one hundred and six cases from the literature of rather more than seventy years. Apart from the fact that many cases remain unpublished, in this country at all events—and of such I have myself seen three, and heard of several more—the attention which has recently been drawn to the subject by the monographs already mentioned has led to the publication since their appearance of a relatively much larger number of cases. Thus, in the six years following Sattler's work, 1881-86, I find records of twenty-five cases.

The aneurismal protrusion of the eye is usually on the one side alone but may be bilateral. Bilateral aneurism occurred in six of the one hundred and six cases collected by Sattler, and three have since been recorded by Secondi, Hoffmann, and Neiden respectively. They form, therefore, probably about seven per cent. of all cases.

The symptoms of aneurism of the orbit are—exophthalmos with pulsation to be felt over the eye, and a more or less continuous murmur, which can be heard over the forehead and eye. On pressure over the protruded eye it can to a great extent be replaced, and on relief of the pressure it immediately returns to its former position. The vessels of the lids are greatly distended, and the lids themselves red. The retinal veins are found on ophthalmoscopic examination to be very greatly dilated, often exhibiting a marked pulsation, while at the same time the arteries are usually diminished in calibre. There may sometimes, too, be found an appearance exactly like neuritis.

The subjective symptoms complained of are pain and noises in the head and ears. The pain may be excessive, and is generally most distressing at an early stage of the affection. The noises appear to be much more troublesome in some cases than in others; they sometimes greatly interfere with sleep. They cease, or are much diminished in intensity, by compression of the common carotid artery, which at the same time causes the murmur and pulsation over the tumour to disappear. When the aneurism, as is usually the case, is suddenly formed, the patient is conscious of a great noise, which is sometimes described as like the report of a pistol or the crack of a whip, in the head.

Aneurism of the orbit may be either idiopathic or traumatic. The latter is caused mainly by severe injuries to the head, which have generally at the same time produced fracture at the base of the skull. A few cases have been the result of penetrating wounds in the orbit. Traumatic orbital aneurism is most common in men, who are more exposed to accidents, than women. Idiopathic orbital aneurism on the other hand, has been met with more often in women than in men. Sattler's statistics give over seventy per cent. in women, and from thirty to fifty as the most common age for its occurrence.

There are a good many different possible causes of aneurismal proptosis, and no doubt most of these have actually happened.

The following have been demonstrated on *post-mortem* examination :—spontaneous as well as traumatic rupture of the internal carotid artery in the cavernous sinus, aneurism of the ophthalmic artery in the orbit, and of the same artery in the cranium. The first is undoubtedly by far the most frequent cause. The effect of the giving way of the arterial wall in this position is to cause an increased pressure within the sinus, and a consequent retardation of the current in the orbital veins, and therefore mainly in the ophthalmic vein. Pulsation, however, as is pointed out by Sattler, probably only begins when, after dilatation of the veins, the blood current is reversed in them, so that arterial blood courses through the dilated ophthalmic vein. On this account it is generally found that some considerable time elapses between the occurrence of the lesion which has caused the rupture and the establishment of a pulsating tumour in the orbit with exophthalmos.

The *prognosis* of orbital aneurism is bad as far as the vision goes when the case is left to itself, but the condition seems rarely to cause death. A few cases have undergone a spontaneous cure, probably from thrombosis which has spread to the sinus, and has eventually even led to a closure of the aperture in the arterial walls.

The *treatment* of orbital aneurism may be divided into medical and surgical treatment. The fact that several spontaneous cures have taken place might well encourage a trial of such measures as are likely to diminish the blood pressure and favour coagulation, before resorting to any more radical interference with the circulation of the parts affected; and indeed by rest, low diet, local or general blood-letting, the application of ice compresses over the orbit, and the internal administration of tincture of veratria, iodide of potassium, &c., good results appear actually to have been obtained. Theoretically it would seem, however, very unlikely that such treatment would be sufficient, except in some cases, probably for the most part idiopathic, where an actual aneurismal dilatation of the artery at the site of its rupture exists. There is good reason to suppose that such cases are comparatively rare, and as a general rule surgical interference is called for in addition to medical treatment. Various methods calculated to lead to coagulation in the vessels of the orbit have been from time to time tried. Of these, the safest is undoubtedly electrolysis. This treatment is not likely to lead to sufficient coagulation in the case of the common cause of aneurism of the orbit, viz., rupture of the carotid in the cavernous sinus, to effect a firm closure of the breach in the arterial wall against an undiminished blood pressure.

It has, however, been successful in some cases of aneurism, and was so in one case which was under Dr. Argyll Robertson's care at the Edinburgh Royal Infirmary, and in which Dr. Duncan operated by electrolysis. The aneurism was the result of a penetrating wound of the orbit, and may therefore have not unlikely been a spurious aneurism of the ophthalmic artery. Such cases, as well as perhaps some others of the more common nature referred to, where at the same time an aneurismal sac has formed at the seat of the rupture, are the only ones likely to be benefited by electrolysis alone, combined with proper medical treatment.

A larger range of usefulness might be expected by combining electrolysis with intermittent or constant digital compression of the common carotid on the affected side. Compression alone, though it has led to a few cures, has on the whole been unsuccessful; still it should always be given a trial before resorting to ligature, and especially would it be advisable to make the trial in conjunction with electrolysis. Ligature of the common carotid has proved successful in rather over sixty per cent. of the recorded cases in which it has been tried. The cure has usually been effected within six weeks after the operation, though in some instances it has been delayed for a good many months. When it fails, the treatment should consist first in digital compression, and afterwards ligature of the other carotid, which has in two cases at least been successful. In cases in which recurrence takes place after ligature of the carotid, it has usually done so very shortly after the operation; and, indeed, as a rule, it is not many hours after the stoppage of this channel that the pulsation and murmur over the tumour make their appearance, even in the cases which are eventually cured by the ligature. Some cases have, however, only recurred after an interval of several months.

INJURIES TO THE ORBIT—BLEEDING INTO TENON'S CAPSULE.—A disagreeable and somewhat alarming *contretemps* occurs occasionally during the performance of tenotomy of the recti muscles for strabismus. All at once the eye is found to begin to protrude, and in a few seconds becomes rigidly fixed, so that if the operation is not completed, it then becomes impossible to complete it, and indeed the lids can hardly be separated. This is due to bleeding into Tenon's capsule. I have observed it altogether about a dozen times, both in my own practice and that of others. Generally, it is owing to the opening made in the capsule being too small for the escape of blood, which, when an unusually free bleeding occurs, may take place into the capsule, from which it cannot be dislodged even if the opening be at once made, as it rapidly passes all round the eye and coagulates. Often at the same time there has been some struggling on the part of the patient, or some difficulty in respiration when an anæsthetic has been given, which has favoured the bleeding. No doubt in the cases in which it occurs a somewhat larger vessel than usual has been cut across, and it is said by some that this is likely to happen if the scissors be made to cut too deeply and too freely near the caruncle. In my own experience, however, I have found it just as liable to happen where the incision is altogether away from the region of the caruncle. No harm seems ever to result from this accident beyond a very black eye. When it occurs the patient should be kept in bed for the day, and ice compresses applied over the eye for some hours until the swelling becomes less tense.

Other cases of effusion of blood inside the orbit are caused by perforating wounds or injuries to the head, which have usually at the same time led to fracture of some of the bones of the orbit. Protrusion of the eye from hæmorrhage into the tissues behind it is a condition which may generally be inferred to be present when the proptosis has taken place suddenly after a severe blow on the head. It is a symptom of great gravity, as even the few cases which have been recorded, where there has been no fracture of the orbit, have ended fatally, while it is almost a certain indication of such a fracture. Sometimes, indeed, there can be no doubt of this, as the blood escapes by the nose or mouth. That which is caused by a perforating wound is not so serious, and generally, if it has not caused an aneurism, becomes absorbed in a few weeks. Besides the exophthalmos caused by the hæmorrhage, there is more or less ecchymosis produced after some time in the conjunctiva and lids, which renders the diagnosis of the cause of the protrusion easy. It appears to be doubtful if spontaneous hæmorrhage into the orbit ever occurs,—at all events it is extremely rare.

Where there is a fracture of the orbit it not infrequently happens that the line of fracture passes through the optic foramen, and thus, by laceration of the nerve or hæmorrhage into its sheath, gives rise to sudden blindness. Fractures of the margin of the orbit alone have been met with, but are rare accidents.

FOREIGN BODIES IN THE ORBIT.—Foreign bodies of different sizes may become lodged in the orbit. Often this accident happens without the patient being aware that anything has entered the orbit. For instance, it may result from a fall on some sharp piece of wood or branch of a tree which has broken off and remained embedded in the orbit, but not observed owing to the patient being at the same time stunned. At other times it is caused by the breaking off of a knife or some other weapon with which the patient has been accidentally or intentionally wounded in this situation. Several foreign bodies even may be lodged in the orbit, as the result of some explosion.

FIG. 106.—Piece of clay pipe stem removed from the orbit.

Fig. 106 is a drawing of a piece of a clay pipe stem which I removed from the orbit of a young man some years ago. It was deeply embedded in the orbit, where it remained for ten days, without causing an abscess or in any way damaging the eye. Curiously enough I have seen two other cases of the same accident.

The possibility of a foreign body being present should always be borne in mind in connection with the treatment of cases of penetrating wounds of the orbit. An accident of this nature is not so very uncommon, and most frequently the foreign body is situated to the inner side of the orbit. It causes exophthalmos, the eye at the same time being displaced in accordance with the position occupied by the foreign body.

The *diagnosis* may be difficult, but can generally be made by careful probing. The *treatment* then consists in removing the obstacle, which usually, unless there be others present as well, soon leads to healing. Sometimes the injury causes death. When this is the case, it is generally either owing to the direct wounding of the brain, or to hæmorrhage.

GRAVES' DISEASE.—The main symptoms of this disease are—(1.) Rapidity of the heart's action; (2.) Enlargement of the thyroid; (3.) Protrusion of the eyes. The association of the two last symptoms have led to the name *exophthalmic goître*, which is frequently used as synonymous with Graves' disease. In Germany it goes by the name of *Basedow's disease*, Basedow having undoubtedly been the first to give a complete description of the characteristic symptoms, and more especially to direct attention to the connection between the exophthalmos and the other symptoms. But the same disease had been known several years before in this country through the teaching of Parry and Graves, who, however, did not lay particular stress on the exophthalmos, though the latter at any rate fully appreciated the connection which existed between the other symptoms.

All these symptoms are not invariably met with in Graves' disease; thus there may be palpitation and goître alone, or with hardly any appreciable protrusion of the eye, or the palpitation may exist along with proptosis alone, the thyroid being not appreciably enlarged.

The increased rapidity of the heart's action is the most constant and essential symptom, so that although exophthalmos from Graves' disease may exist without any appreciable cardiac acceleration, the diagnosis of the true nature of the protrusion of the eyeballs under such circumstances is difficult or altogether uncertain. In most cases the heart beats amount to from 100 to 160 in the minute, but they are sometimes more frequent. I have counted in several cases over 200 beats a minute. The pulsation is felt very distinctly over the carotids, which are most frequently what have been called "hammering carotids." They, as well as other arteries of the head, are dilated. The veins in the neck are also dilated, and on auscultation over them a hæmic murmur can generally distinctly be heard.

The goître has usually a soft elastic feeling, and communicates to the hand either a very distinct pulsation or a thrill which is synchronous with the systole of the heart. The enlargement varies very much in different cases; it is rarely extreme. Not infrequently the one lobe of the thyroid, and almost invariably the right, is more enlarged than the other; while in a few cases the one lobe alone has been found to be enlarged. Hughlings Jackson found the right lobe larger than the left in eight consecutive cases which he had seen, and in which he had paid attention to this point. Cases are on record where death has occurred from dyspnœa, caused by the pressure of the enlarged thyroid on the trachea. In other cases again it has been found necessary to remove the isthmus to prevent this occurrence.

The exophthalmos is almost always bilateral, though not infrequently one eye is protruded more than the other. Occasionally it is altogether confined to one side. This I have seen in two cases, one of which was a man of fifty, and the other a woman of forty. The protrusion of the eye takes place directly forwards, and in most cases, and always where the disease has not existed for any great length of time, the eyes may be pressed back into the orbit without much difficulty, but regain their former position on the relief of the pressure. A hæmic murmur may be heard on auscultation over the eye. The degree of protrusion varies very much. In many cases it goes on increasing slowly or rapidly for some time, and then again becomes less; in others it remains pretty much the same, though subject to slight differences, according to the patient's state of health. It may be so excessive as to render it impossible for the lids to cover the eyes, or even cause the eyes to become dislocated out of the orbit. Such a degree of exophthalmos is, however, very exceptional.

The protrusion does not of itself cause any interference with the movements of the eye. In exceptional cases these movements are impeded by complication with paralysis. Thus Bristowe has recorded one case where there was at the same time ophthalmoplegia externa. I have seen with Dr. Smart of Edinburgh a case complicated by bilateral abducerus paresis. Other forms of paralysis of the ocular muscles have also been met with in Graves' disease, a circumstance which is interesting in connection with the probable pathology of the disease.

The exophthalmos of Graves' disease is in most cases accompanied by a frightened staring appearance, which is very characteristic, and due to the greater than normal retraction of the upper lid. This involuntary widening of the lid aperture, caused by the contraction of the unstriped muscular fibres of Müller, seems first to have been described by White Cooper, but now generally goes by the name of "Stellwag's sign," as Stellwag succeeded in directing general attention to it. Stellwag pointed out at the same time that in cases which exhibit this symptom there is a more or less complete absence of involuntary blinking. This retraction of the upper lid is not met with in other forms of exophthalmos, and is therefore a point of diagnostic importance in the cases where exophthalmos is confined to one side, or not accompanied in a marked degree by the other symptoms characteristic of Graves' disease. It frequently exists at a time when there is little or no protrusion of the eyes, and calls attention to the condition of the circulation and thyroid. An appearance very similar to Stellwag's sign is produced by cocaïne dropped into the eye.

A much more important, though, so far as my experience goes, less frequent symptom, is that which is called "Graefe's sign." This consists in the loss of association of the movements of the upper lid with the eye. Thus, when the eye is directed downwards, the upper lid either does not follow it at all, or more frequently moves along with it for a certain distance and then remains stationary. Graefe's sign is also highly characteristic of Graves' disease, and is not found at all, or only extremely rarely, under other circumstances. It may be seen just as the retraction of the margin of the upper lid, before proptosis takes place, or may occasionally first make its appearance later. It may also not be present during the whole course of the disease, but disappear after having existed for some time. It is absent in some cases in which Stellwag's sign is well-marked.

Notwithstanding the presence of these characteristic symptoms which have just been described, there is no defect in the closing of the lids, the voluntary movements of which are retained just as in health, so that it is only when the proptosis has become very excessive that the eyes cannot be properly covered. In such cases, and even it would appear in some cases in which the lids can still be made to afford a sufficient covering, a kind of neuro-paralytic keratitis takes place, which is extremely likely to cause destruction of the cornea. In this way, but apparently only in this way, is the sight endangered by the disease; but such cases are also the ones in which the prognosis is serious for the life of the patient as well.

Besides the main symptoms of Graves' disease, which have now been described, there are other more or less constant ones. One, which in women at all events is often most marked, is an excessive degree of nervousness. The patient is easily startled and frightened, and very readily blushes or is subject to attacks of perspiration. In some of the worst cases rapid emaciation also takes place. More or less anæmia is generally present at the same time, and this may in women be accompanied by menstrual disturbance, but I have seen several cases in women who were not only florid, but appeared to be in every way in excellent health. Some dilatation and even pulsation of the retinal arteries is found in some cases, though in my own experience pulsation at any rate is much less frequent than is assumed by some writers on this subject, and than might perhaps be expected from the evident want of tone in the arteries of the head and the excited state of the heart's action. Hill Griffith, who specially looked for pulsation, in thirty-two cases did not meet with it once. A certain degree of anæsthesia of the cornea is to be found in some cases, and in such there is probably a greater danger of ulceration than in others. This symptom therefore deserves special attention.

Graves' disease is very much more frequent in women than in men. I find from a comparison of the different statistics on this point that quite eighty-five per cent. of all cases occur in women. Possibly, indeed, the percentage may be higher, as the cases in men are perhaps more likely to come under the observation of the specialist, both on account of their rarity and from the fact that the disease appears to be more severe in them than in women. More than half of the cases in women occur between the age of puberty and thirty, a very few before puberty or after the climacteric period. In men, although cases are also met with in young individuals, the average age for the disease to make its appearance is between thirty and fifty.

The course of the disease is usually very chronic. It may last for years in much the same condition, or occasionally undergo an improvement in one or more of its symptoms. After continuing in this way for some time, the symptoms gradually in great measure subside, though frequently some enlargement of the thyroid and often a not inconsiderable degree of exophthalmos permanently remain. Again, a number of cases never completely recover.

On this point I may quote the opinion of Bristowe, that in many cases "the proptosis and goître subside in a greater or less degree, and the palpitation diminishes; while, nevertheless, the patient remains delicate, and with the liability to suffer under excitement or accidental ill health from more or less severe recurrence of palpitation and other symptoms which attend exophthalmos." In a not very large proportion of cases death takes place, either from exhaustion or organic heart disease; rarely, as has been said, from dyspnœa, owing to the pressure of the enlarged thyroid on the trachea. Practical recovery, however, is the most frequent result. Thus Hulke, in opening the discussion on this subject at the Ophthalmological Society, remarked—"It is the most definite and striking example which we can find of a severe and protracted malady which, despite its severity and persistence, has yet a natural tendency to recover. If the patient can survive a certain time, apparently the recovery is a matter of course. We have no cases which are indefinitely progressive."

Graves' disease seems to occur with pretty equal frequency amongst the white races all over the world. Little seems to be known as to its occurrence in coloured races. Dr. Brockman of Madras says—"It is remarkable that although goître is met with in India in great numbers, I have never met with a case of exophthalmic goître among the natives during my fifteen years' practice in diseases of the eye."

The frequency of the disease in women, and its occurrence mainly during the child-bearing period of life, has led many to believe that Graves' disease is intimately connected with affections of the reproductive organs. The occasional occurrence of the disease in women at other times of life, as well as in men, and the absence frequently of any menstrual disturbance, would, on the other hand, point to such a connection not being very intimate. In these cases, too, in which derangements of menstruation are observed, they may well be ascribed to the condition of anæmia, which is so frequently present in this disease. Unmarried women seem to be more subject to the disease than married, and the pregnant condition is supposed by many to exert a favourable influence on it. In one case which I know of, the exophthalmos and the symptoms of Graves' disease were very marked during ten years in the case of a married woman who had had several children, but greatly improved when after that interval of time she again became pregnant. Such cases, however, seem rather to point to the pregnancy as well as the disappearance of the disease occurring both as the result of an improvement in general health, rather than to any direct favourable influence of the one condition over the other. And, indeed, cases are on record where the disease has become markedly worse during pregnancy, just as that state is one not always associated with good general health. Begbie considered albuminuria to be frequently connected with Graves' disease, but this has not been confirmed, the cases where this connection exists being decidedly uncommon. Lately, another affection—viz., acute rheumatism—has been mentioned by West as a frequently associated one. A history of rheumatic fever was got in eight cases out of thirty-eight recorded by West, which certainly does not appear to be a very large proportion, considering the very great frequency of rheumatism, and the fact that exophthalmic goître mostly affects individuals who are delicate in some way or other. Finally, a few cases of bronzing of the skin have been observed in the same connection. Apart though from more or less marked forms of nervousness or nervous affections, there cannot truly be said to be any disease very closely associated with it.

The *pathology* of Graves' disease is still very obscure. Post-mortem examination in cases which have ended fatally has done very little to clear up the difficulty. Some change in the orbital fat—hypertrophy, enlargement of the vessels, &c.—has generally been found. There can be no doubt that both the enlargement of the thyroid and the exophthalmos are originally due to engorgement of vessels, and it is not unlikely again that after long continuance of the process some hypertrophy of the tissues may take place. Still, the protrusion of the eye is mainly a vascular one. Changes have been described in the sympathetic trunk in the neck or in its sheath, but these have apparently been more fancied than real, and at all events have not received very general confirmation. Cheadle found congestion of the pons and medulla in several cases, and Hughlings Jackson has recently recommended a careful microscopic examination of these parts of the brain in all cases where a post-mortem examination is obtained. The hypothesis of a disease of the sympathetic being at the root of the symptoms received the support of Begbie and Trousseau amongst others, and more recently of Eulenburg, Guttmann, and other authorities. Besides, however, the absence of any demonstration of this connection from the pathological side, there are many considerations which render it improbable. In the first place, whilst some of the symptoms might well be taken as evidences of paralysis of sympathetic fibres, there are others which could only be referred to irritation of these fibres. It is unlikely, however, that such an irritation should exist for such a long time as would have to be inferred from the protracted nature of these symptoms. The absence of dilatation of the pupils is also very much against the correctness of such an assumption. Another, too, to which attention has mainly been drawn by Sattler is that neither the goître nor the exophthalmos, which are such prominent symptoms in the disease, can be explained by a paralysis of the vasomotor nerves in the sympathetic. He says —"It has not been experimentally shown that section of the sympathetic has given rise to goître or exophthalmos, nor are there any clinical facts on record where a true paralysis of the sympathetic, though it has produced distinct vasculo-thermal changes, has been followed by either of these symptoms." Sattler considers, therefore, that the symptoms point rather to "a circumscribed lesion in the vasomotor centre, or in some portion of the brain which is situated even more centrally, which directly governs the vessels to both the thyroid and orbital tissues." The acceleration of the heart's action is satisfactorily explained on the assumption of a lesion at the vagus centre, which in this way interferes with its inhibitory action. The frequent greater enlargement of the right lobe of the thyroid would appear also to support this view, as the right vagus has been shown in some animals to be more strongly inhibitory in its action on the heart than the left.

There are many other circumstances in connection with the disease which point too, directly, to a central pathology, and of these perhaps none more strongly than Graefe's sign. It must be remembered that this, though often associated with retraction of the upper lid, is not always so, and in any case the action of the little muscle which brings about that retraction can hardly be strong enough to counteract that of the orbicularis. The symptom is therefore, in all probability, as Sattler has pointed out, an evidence of some central interference with the co-ordinated action of the lid and muscles of the eye. In some cases more striking instances of such defects of co-ordinated movements have been met with. Bristowe and others, for instance, have seen cases where there has been ophthalmoplegia externa. Altogether, then, while the pathology still remains obscure, there is very good reason to assume that the cause of Graves' disease will be found in some central lesion. Whether that lesion, though probably occupying mainly the same site, is always of the same nature, or whether it differs in the curable and slight cases from that which gives rise to the severe and more permanent ones, is a point in connection with which, in the absence of any definite proof of its existence at all, it would be useless to conjecture. There are many points in connection with Graves' disease, too, which suggest a not very distant relationship to hysteria.

Treatment.—There is certainly nothing which can be said to approach the nature of a specific remedy for Graves' disease. Many things have been tried: iron, iodine, belladonna, veratria, bromide of potassium, and other drugs; but though all have had their special advocates, their direct beneficial effects on the disease cannot be regarded as more than doubtful. Veratria, which by some has been looked upon almost as a specific, I have found absolutely without effect; it does not even, in my experience, greatly diminish the rapidity of the heart's action. The most important indications for treatment are—freedom from excitement and worry, and change of air. If drugs are used at all, iron and bromides are perhaps the best; iodine, too, may be used as an external application to the goître. A few cases have been apparently improved by removal of the goître, but except where it endangers life by pressure on the trachea, such treatment would not be justifiable. Recently Hack claims to have cured a case by cauterising the middle and inferior turbinated bodies with the galvano-cautery, and argues from this effect—which, however, has not yet been confirmed by others—that the swelling of the retrobulbar fat, which gives rise to exophthalmos, is in reality a neurosis proceeding from the irritation of the nose. In cases where the protrusion of the eye is excessive, von Graefe recommended tarsoraphia, or a shortening of the lid aperture. The operation is certainly useful, but where ulceration of the cornea has already begun it is better to bring the lids more completely over the cornea, either by paring their edges for about two-thirds of their extent in the middle, and causing them to grow together by uniting them with sutures, or, what does equally well, merely keeping them closed by loops of horse-hair in the manner described in the chapter on Operations.

Enophthalmos, or the sinking back of the eyeballs into the orbit, exists as a consequence of the disappearance of the orbital fat in very old people. It is also met with in some cases where there has been fracture of the bones of the orbit. It has occasionally been observed, too, apparently as a rare vasomotor affection, but little is known of the pathology of this form.

SHRINKING OF THE ORBIT occurs sometimes when the eyeball has been removed in early childhood. The diminution takes place mainly, if not entirely, by approximation of the roof and floor of the orbit, while the sides retain their normal dimensions.

CHAPTER XIV.

AMBLYOPIA, AMAUROSIS, AND OTHER ANOMALIES OF VISION.

In this chapter are discussed various alterations of vision which are not accompanied by any very definite objective signs. For instance, we often meet with cases of either complete blindness (*amaurosis*), or more or less defective vision (*amblyopia*), which do not arise from any lesion of any part of the eye itself. Possibly in some cases there may be pathological changes in the eye, but of a nature which the means of examination at our disposal does not enable us to detect with certainty.

The diagnosis of the cause of an amblyopia or amaurosis is often difficult enough. A careful study must be made of the subjective symptoms so far as they can be elicited. To some extent too, we are guided by the other co-existing conditions, as well as by our experience of physiology and of post-mortem examination.

In some cases the colour vision may be defective, in others a particular portion of the field of vision may be lost or be functionally inactive. In others, again, the light sense may be abnormal. Under this category come cases of night blindness, in which the visual acuity may be normal in daylight, but defective so soon as the illumination of objects looked at is reduced beyond a certain degree.

A purely subjective sensation of light (*photopsia*) or of colour (*chromatopsia*) may be complained of from different causes; or black spots, the so-called *muscæ volitantes*, may be seen floating, more or less constantly, in front of the eyes.

Under the heading of amaurosis and amblyopia used to be included many affections, the causes of which are now apparent since the invention of the ophthalmoscope has rendered it possible to make a complete examination of all parts of the eye. Such cases are described under the diseases which are now known to cause the blindness, and are not referred to here.

CONGENITAL AMBLYOPIA.—Defective vision, when congenital or originating early in life, often escapes observation, unless the degree is considerable, until the difficulties experienced at school attract attention. The higher degrees of amblyopia are mostly associated with nystagmus. It is by no means an uncommon thing to find very considerable amblyopia and nystagmus without any marked objective cause, either in the dioptric or percipient parts of the eye. In such cases there is usually a history of early severe inflammation of the cornea, followed by dense opacities, which have slowly cleared away, leaving little or no trace; or of some cerebral affection, which has interfered, more or less completely, with the visual centres, and which has been followed by only partial recovery. In both cases the conditions have been unfavourable to the acquirement of that superiority of the vision of the centres of the retinæ, or foveæ centrales, over the other parts, which characterises more normal vision, and admits of the usual steadiness of fixation.

Congenital amblyopia, ranging from the slightest defects of vision to that where the acuity is but one-tenth or less of the normal, and unaccompanied by nystagmus, is very frequently met with without there being any objective sign or any history to account for it. Frequently, too, the degree of amblyopia where there is some defect is much greater than can be accounted for by the defect alone. When amblyopia is present only in one eye, it is still more likely to pass unobserved, unless, as is frequently the case, it gives rise to strabismus, owing to the existence of other predisposing causes. Often a unilateral congenital amblyopia is only accidentally discovered late in life, when anything, some trifling accident to the eye, it may be, has drawn the individual's attention to it.

SIMULATED AMBLYOPIA.—Amblyopia is sometimes simulated. In this country it is rare, comparatively speaking, to meet with this, and the deception is not so well carried out as a rule that there is much difficulty in detecting it. In other countries, where there is a compulsory military service, simulated partial or complete blindness is very common.

There may be a simulation of either bilateral or unilateral defect of sight. The latter is by far the more common, and fortunately, too, it is the more easily detected. It is mostly an amblyopia, and not a complete blindness, of the one eye which is feigned, so that the diagnosis may not be at all easy. When bilateral amblyopia is simulated, a little ordinary care on the part of the individual is all that is required to make it almost impossible for one to detect it with certainty. Yet it is strange how often some inconsistency between the admitted acuity of vision found on examination for distance and for near, or in connection with the glasses which are admitted to effect some improvement in vision, affords a clue to the character of the amblyopia. I have, for instance, repeatedly, and more especially in young girls, obtained very considerable improvement of vision by the use of a convex and concave glass, which exactly neutralised each other. It requires on the other hand a very great amount of care, and considerable knowledge of the subject, to simulate well monocular amblyopia. The certainty of the diagnosis depends pretty much, too, on the degree of the pretended blindness.

A great number of tests have been devised for the detection of this kind of deception, all of which it is probably possible to elude. An easily applied test is that which can be made with Snellen's coloured letters. These are transparent red and green letters of different sizes. The patient is first made to read them out without anything before his eyes. A pair of reversible spectacles, one eye of which is of green glass, and the other of red, is then put rapidly upon his nose, care being taken that the eyes are all the time kept open. As the green glass entirely excludes all the rays from the red letters, and the red all the rays from the green, some of the letters are visible only to the one eye, and others only to the other. Any mistake made in the reading of the letters is in this way easily detected. The slightest blinking of one eye is sufficient to show one with which eye each particular letter is seen, so that with care it is possible for a clever deceiver to avoid falling into this trap, although the manner in which the individual behaves may be generally sufficient to arouse or confirm one's suspicions. Another simple test in cases where complete or almost complete blindness of one eye is simulated consists in holding a prism with its base directed upwards or downwards in front of the good eye. If this produces diplopia, there is evidently vision in both eyes; but the object of this test again is of course easily defeated by any one denying that he sees double. An important modification of the test is made by Alfred Graefe. The individual tested is made to shut the blind eye, and the prism is held in front of the good one, in such a position that some of the rays pass into the pupil through the prism and some directly. This causes monocular diplopia. The patient, thus thrown off his guard, often admits that he still sees double when the blind eye is uncovered, and the position of the prism altered to the slight extent required to intercept all the rays passing into the pupil of the seeing eye, under which circumstances the diplopia is of course binocular, and its continuance is a proof of vision in the eye said to be blind. A good plan in cases of simulated monocular amblyopia, and one which at the same time affords some test of the degree of visual acuteness in the eye said to be defective, is to cause the patient to read through a strong convex lens (+5.0 or +6.0, if it be emmetropic), and then by slowly withdrawing the print beyond the point at which it can be distinctly read through the lens. If under these circumstances the patient still continues to read he must be doing so with the eye which he asserts to be amblyopic. Von Graefe's test of placing a prism, with its base directed inwards or outwards, in front of the seeing eye, and observing whether a lateral movement of the eye is thereby induced, is perhaps the best test for simulated amaurosis of one eye, because where binocular vision exists such a movement almost always takes place involuntarily owing to the unconscious desire for the fusion of the two images.

AMBLYOPIA AND AMAUROSIS DUE TO CHANGES AT THE VISUAL CENTRES, &C.—A good number of cases of amblyopia and amaurosis occur more or less suddenly, and unaccompanied, for the time at least, by any ophthalmoscopic changes. In such cases the diagnosis has to be made from a consideration of the history. When the blindness comes on in the course of a severe illness, or during the period of convalescence—for instance, in scarlet fever, puerperal albuminuria, &c.—the cause is more often uræmic, and the prognosis favourable, more especially if the pupils continue to respond to light.

Amblyopia or amaurosis occurring after severe hæmorrhage is very unlikely to be recovered from, and is generally followed some time after by optic atrophy.

The *prognosis* is even worse in cases of unilateral amaurosis after a severe fall on the head. These cases are of pretty frequent occurrence, and are probably almost invariably due to laceration of the optic nerve, or hæmorrhage into the sheath of the nerve, owing to fracture of the roof of the orbit, the line of which fracture usually passes through the optic foramen. The atrophy to which this lesion gives rise is not visible ophthalmoscopically until after some time.

The blindness which one meets with in young children after severe cerebral symptoms is often unaccompanied by ophthalmoscopic changes, and is due no doubt to interference with the functions of the visual centres. It is often partially recovered from, but even in cases where the pupils remain active I have seen permanent blindness remain.

The rare cases of blindness from lightning do not seem to have received a proper explanation as yet. Only one case of this nature has come under my own observation, and that after complete recovery. In this case, which was kindly brought to my notice by Dr. Burn Murdoch of Edinburgh, there appears to have been complete amaurosis, lasting for six weeks, with ptosis. On the whole, judging from the recorded cases, the prognosis seems favourable.

In cases of lead poisoning very considerable blindness is sometimes produced. I have seen the blindness so great that there has been doubtful perception of light, and yet recovery take place. Some of the symptoms in these cases, more especially with respect to colour vision, are often very similar to what is met with in hysterical amblyopia. As in that form of amblyopia, too, there seem to be considerable differences in the nature of the visual defect, and also a good deal of inconstancy about the symptoms of any particular case. Amaurosis from lead poisoning, when unaccompanied by ophthalmoscopic changes, appears most frequently to be transient. It is one of the rarest symptoms of lead poisoning, and although different explanations have been offered the true nature of the blindness is unknown. Most probably the view recently put forward by Günsberg is the correct one. According to this view the amaurosis is not a direct effect of the lead on the brain, but is due to a co-existing uræmia.

Central Toxic Amblyopia.—The symptoms of this rather common affection are—gradual failure of sight to much the same extent in both eyes; absence of any restriction of the field of vision, or indeed of any interference with the functions of the peripheral portions of the retina; and the existence of an oval scotoma, most marked for colours, extending from the point of fixation, which it involves, to the blind spot. Most of such cases are met with in men after the age of forty. Occasionally younger men are affected; the youngest undoubted case I have seen was twenty. This form of amblyopia is very rare in women, though not quite so rare as was at one time supposed.

Those who suffer from it are almost invariably smokers; and generally they have smoked for many years before becoming affected. It is very doubtful if alcohol has anything to do with it, though this is maintained by some. Certainly one not infrequently finds that those affected not only smoke, but also drink to excess; yet the same symptoms are never observed to follow the excessive use of alcohol alone, though they are very frequently met with in smokers who abstain completely from alcohol in any form. As a rule, when the affection begins, no change has been made recently in the amount smoked, and this circumstance accounts for its being comparatively rare to find that patients suspect the cause of their amblyopia. Indeed, they not infrequently begin to smoke more after their sight has begun to fail, on account of the worry that this causes. Very often smoking is indulged in on an empty stomach, the first thing in the morning or very late at night, or, in the case of bad sleepers, during the night. It is owing to this circumstance, probably more than to the difference in the kind of tobacco smoked, that tobacco amblyopia is more common in the working than in the educated classes. Many men have to rise early and work several hours before partaking of a substantial meal, but nevertheless smoke during this time.

The fact of the toxic effect of the tobacco getting at some particular time the upper hand, although individuals have smoked as much and often more at other times, and continuously for years, points to there being some recent diminution in the power of resistance. What this has been it is not always easy to find out. Sometimes their has been some slight indisposition or dyspepsia, sometimes loss of blood, at other times merely sleeplessness, or sleeplessness caused by anxiety and trouble. In many cases it can only be ascribed to want of nerve tone, or some senile loss of vigour. A frequent cause no doubt is the undermining of the system produced by alcoholism, so that alcohol is thus indirectly a factor in the etiology. When any such predisposing cause exists, then smoking at a time when there is no counter stimulus of the food renders the poisoning action much more likely to take effect. According as such causes, too, are more or less pronounced, and dependent no doubt as well upon the individual tolerance of tobacco, we find in different cases of this form of amblyopia that the patients have been in the habit of smoking very different amounts of tobacco. As a general rule not less than three to four ounces of strong tobacco are smoked in the week, often much more—in exceptional cases less. In women a smaller quantity seems to suffice to bring on the symptoms. The same symptoms are produced by chewing tobacco.

It would be strange indeed if tobacco were the only poison which gives rise to central amblyopia. Cases do occur in which it is doubtful whether tobacco is the cause, as, for instance, when the amount consumed is very small, or where the blindness appears to have come on some time after smoking has altogether been given up; but, with the exception of bisulphide of carbon, which produces much the same symptoms, only, as far as my experience goes, relatively greater amblyopia with relatively less marked colour defect, the evidence in favour of other poisons is as yet very far from conclusive. Some cases of retrobulbar neuritis very closely resemble toxic central amblyopia, more especially when the defect produced by that inflammation is pretty similar in both eyes.

Although the bad effects which tobacco may produce on the vision have been long known, it is only comparatively recently that cases of this nature have been diagnosed with certainty, and the cause of the affection properly understood. This has been mainly owing to the manner of testing and defining the limits of the scotoma by means of colour tests. The defective area is oval in shape, with its long diameter horizontal, and stretches from the outer side of the blind spot to very slightly to the inner side of the point of fixation. It corresponds to the external projection of that portion of the retina which lies between the optic nerve and the outer margin of the macula, a portion which recent investigation has shown to be supplied by a special bundle of optic nerve fibres—the so-called papillo-macular bundle. The form sense as well as the colour sense is defective over this area, but the patient is not conscious of a limited defect—that is, it does not appear in the form of an oval dark spot in the field of fixation, or, in other words, the scotoma is not *positive*, but what is called a *negative scotoma.*

Practically all that is required to diagnose a central scotoma is to cause the patient to fix some object, preferably on a dark background, a foot or two from his face, and then to hold a small piece of coloured paper first an inch or two to the outer side, and then a similar distance to the inner side of the point of fixation, when it will be found, if the scotoma be marked, that the colour is not recognised in the first situation, but is immediately recognised in the second. As the defect is most marked for red and green, only these colours need to be used, unless, as I have several times seen, there should happen to be at the same time congenital red-green blindness, when blue and yellow must be used instead. The limits of the scotoma can be mapped out with the colours, but for this purpose smaller squares should be used, and the shade not taken too bright. By using very small white objects at a greater distance the corresponding form-sense defect in the scotomatous area may usually readily be detected; that is to say an absolute scotoma for white is also to be found. When the scotoma is not very pronounced, and the visual acuity consequently not greatly reduced, there is more difficulty in demonstrating its existence: it will then be found that, although the ordinary colour of the test square is recognised to the outer side of the point of fixation, it appears more vivid and distinct when removed to the inner side. By using light shades of red and green in such cases the defective colour sense can be demonstrated with certainty.

As it is only over a particular area that the colour sense is defective, the patients are often not aware of the colour blindness they have acquired: a large surface of red or green appears to them quite as distinct as formerly. By taking advantage of this a pretty rapid method of diagnosing the central scotoma may be practised. If the patient be made to fix a white mark on a large surface of red paper on which is painted a green spot of a hue exactly complementary to the red, and of as nearly as possible the same shade, he is quite unconscious of the green, and considers the surface to be uniformly red when the spot falls within the area of the scotoma, while he immediately sees it in its proper colours if it be held so as to cause the spot to fall on a healthy portion of the retina.

Individuals with tobacco amblyopia often complain of seeing worse in a strong light. This is owing apparently to some co-existing hyperæsthesia of the retina, which causes any glare to be uncomfortable. They see white letters on a black background better than the ordinary black letters on a white ground.

The *prognosis* in tobacco amblyopia is on the whole very good. A large proportion of cases completely regain vision if the tobacco be stopped. Improvement, though less rapid, generally takes place when the amount consumed is reduced, or when the quality of the tobacco used is milder. This is the more likely to take place if smoking be indulged in only after meals. Sometimes, though very rarely, recovery may take place without any change of habit with respect to smoking. When this happens it is probably to be explained on the assumption that some condition of the system, which has permitted the tobacco to get the upper hand and produce its poisonous effects, has been so far recovered from as to enable the individual to throw off the poison, notwithstanding the cumulative action which has taken place. It is not probable that the symptoms have been due to anything else. A very varying time elapses between the stopping of the tobacco and complete recovery, depending partly on the degree of blindness and partly on individual peculiarities. Often there is a period of a month or six weeks, or even longer, before improvement begins at all.

Probably in many cases where the vision is apparently reduced to much the same extent, there may yet be great differences in the density of the scotoma, and consequently in the extent of the toxic action which has taken place. On the whole, the younger and more healthy the individual, and the smaller the degree of blindness produced, the more rapid is the cure likely to be.

The *treatment* consists in stopping the use of tobacco in any form altogether. Although merely diminishing the amount consumed may be sufficient, it is found, as a rule, easier, and it is certainly safer, to give it up altogether. In addition, either inhalation of a few drops of nitrite of amyl or the internal use of nitro-glycerine in small quantities ($\frac{1}{200}$ gr.) sometimes appears to hasten recovery. Attention must also be paid to the general health, and an attempt should be made to improve any conditions by which the system is likely to be lowered.

The lesion which produces this peculiar form of amblyopia is not known. The regularity in the shape of the scotoma, as well as the complete recovery which so often takes place, render it extremely unlikely that, as has been maintained by some lately, it is a form of retrobulbar neuritis. In undoubted cases of that nature, although the central vision is mostly affected, there is rarely either a similar amount of blindness in both eyes, or any constancy in the shape of the scotoma, while at the same time there is not infrequently some slight peripheral restriction of the field, and rarely as complete, and certainly by no means as frequent, recovery. It is more probable either that the poisonous effect is exerted on some part of the brain which includes the central terminations of the papillo-macular fibres, or that some limited vasomotor change affects these fibres in some part of their course. Apart from the cases of true retrobulbar neuritis referred to at page 233, there are others which exhibit the symptoms of a central scotoma, but which, nevertheless, are not, correctly speaking, either cases of central or of toxic amblyopia. It is the existence of such cases, no doubt, which has led to the mistake of considering toxic amblyopia to be due to inflammatory changes in the optic nerve. The true cases of toxic amblyopia have not so far been examined anatomically.

An attempt has been made recently by Jensen to classify these cases of atypical central amblyopia in which the appearance of a central scotoma in one or both eyes is only a transitory phenomenon, and not the essential and only characteristic symptom of the affection. Jensen's statistics are taken from Hansen-Grut's clinic. Including one group of retrobulbar neuritis, about 30 per cent. of the cases of central scotoma examined, which numbered in all considerably over 400, were considered to be atypical. Jensen classifies these atypical cases under the following heads:—Stationary scotomatous optic atrophy; progressive scotomatous optic atrophy; bilateral optic neuritis with central scotoma; unilateral amblyopia, neuritis or atrophy with central scotoma; and glaucoma simplex.

The most important of these groups is the *stationary scotomatous optic atrophy*. Cases of this nature were found to constitute about 25 per cent. of the atypical varieties of central amblyopia, or 7 per cent. or 8 per cent. of all the affections characterised at some time or other in their course by a central scotoma. Of this group Jensen gives the following description:—

"It occurs exclusively in young men under the age of 34, usually between the twentieth and twenty-fifth year; occasionally showing a hereditary tendency, sometimes apparently caused by want of sleep and other weakening factors; often without any demonstrable cause. The affection begins with considerable amblyopia, which occurs either suddenly or reaches a maximum in the course of a short time, without being accompanied by disturbances of general health; it develops usually simultaneously in both eyes.

"On examination of the field of vision a central scotoma is found of about the same size and form as in amblyopia centralis, but much more complete—white objects, presenting a visual angle of 1 to 2 degrees, disappearing either entirely or becoming very indistinct within its area. Corresponding to this, fixation is uncertain or eccentric. During its course the density of the scotoma, and consequently the central amblyopia, remains as a rule unchanged.

"The periphery of the field of vision may present slight anomalies in the colour sense; rarely complete or permanent red-green blindness. Further, transitory defects of peripheral vision for white objects may arise. As a rule, the periphery remains normal during the whole course.

"Ophthalmoscopically there is found to be complete atrophy of the papillæ, and this sometimes very early; as a rule, it is decided before the lapse of a year. Occasionally there may be a suspicion of a neuritic origin; on the other hand, a decided intraocular neuritis is never found to precede the atrophy. The prognosis of this affection is bad *quoad restitutionem*, but absolutely good *quoad cæcitatem*."

Progressive scotomatous atrophy, which occurs with about the same frequency as the stationary form, is met with only in men of middle age, rarely if ever before 35. It is to be regarded more as a particular form of development of spinal atrophy than an independent disease like either central toxic amblyopia or stationary scotomatous atrophy. There is frequently a syphilitic history, and an association with more or less well-marked premonitory symptoms of tabes dorsalis. The blindness usually comes on in the one eye before the other. The main points in connection with these affections are thus summed up by Jensen:—

The affection begins as a central amblyopia, with a central colour scotoma and intact periphery.

The scotoma has exactly the same form and degree of saturation as that in amblyopia centralis, and, just as is the case in that affection, the defect for white objects can only be discovered for minimal visual angles. During the course of the disease the scotoma retains its size and its relative character.

In the periphery, on the other hand, anomalies come on characteristic of progressive atrophy, the boundaries for colours become narrowed centripetally; and finally, though often very late, those for white also. At this stage the original scotoma is frequently unrecognisable; the disease runs a course similar to an ordinary progressive atrophy, and the prognosis is as bad as other cases of that description.

Complete atrophy of the papillæ comes on with certainty, and as a rule earlier than in the preceding form; it may generally be diagnosed at an early stage.

REFLEX AND HYSTERICAL AMBLYOPIA.—The forms of blindness which may be included under this heading are, for the most part, cases of what, in contradistinction to central amblyopia, might well be called *peripheral* amblyopia. They are perhaps in many respects the most interesting and curious cases of amblyopia without ophthalmoscopic changes. Reflex irritations may proceed from the teeth, stomach, or intestines, or from the uterus and ovaries. The amblyopia to which the irritation gives rise, after persisting for months, often disappears quickly on removal of the source of irritation. Very little is known of the more exact pathology of these cases. They are vasomotor disturbances no doubt, but whether of the retina or of the centres of vision is not very clear. The former appears, however, the more likely, as they frequently exhibit the symptoms of anæsthesia or hyperæsthesia of the retina, the field of vision as a rule being concentrically restricted, sometimes to a very extreme extent, while the central vision is not so very much impaired. A remarkable peculiarity, too, is the inconstancy shown in the degree of restriction of the field of vision when successive examinations are made at short intervals. The only thing that appears to be at all constant is that the field of vision, tested by moving the test object in towards the centre, is larger than when tested by moving it in a centrifugal direction, showing a rapid tiring of the retina, or, it may be, of the attention to visual impressions. In some of the more distinctly hysterical cases, too, there appears to be a considerable improvement in peripheral vision in subdued light.

Cases of complete hysterical amaurosis are the most extraordinary, both in their symptoms and in the suddenness with which, under certain strong emotional excitements, they may be recovered from. The condition is, of course, mostly met with in women, but I have seen with Dr. Byrom Bramwell an absolutely typical example of hysterical amaurosis in a boy of twelve. There is in such cases evidently a withdrawal of the attention from the impressions received by the visual centres, whilst at the same time a more or less conscious perception of the impressions remains. The colour vision in hysterical amblyopia is very curiously abnormal, and often affected in quite a different way from that met with where defects in this function accompany more definite lesions. Thus blue and yellow frequently appear to be less well recognised than red and green, but all sorts of possible varieties in the state of the colour vision occur.

SUBJECTIVE SENSATIONS OF LIGHT AND COLOUR may proceed from some irritation of the retina, or may be due to irritations of the visual centres in the brain. It is by no means always easy to detect in any particular case what is their origin. Generally speaking, photopsia due to retinal irritation is most evident after any prolonged exposure of the eye to strong light, or after anything causing fatigue either of the retina or of the body generally, and tends to become less marked or to disappear altogether when the eyes are rested, and therefore especially at night. Photopsia caused by irritation of the visual centres, on the other hand, is often most distressing at night, and at times when the eyes are not subjected to stimulation from the ordinary objective sources. In some cases of hyperæsthesia of the retina complaints of coloured vision are made. This kind of chromatopsia is evidently the result of the response to stimuli, which would otherwise, on account of their feebleness, be disregarded. Owing, however, to the irritable condition in the retina, or of the more central portions of the visual apparatus, these feeble stimuli evoke sensations, just as hyperæsthetic individuals may be affected with pains for which little or no objective cause can be detected.

A curious form of subjective colour sensation has recently received a good deal of attention. It is mostly met with in aphakia after cataract extraction, and consists in a more or less constant sensation of everything looked at being coloured a vivid blood-red. There is no veiling of the objects, but merely a marked red coloration. The condition, to which the name *erythropsia* is generally given, has not yet received a satisfactory explanation. It has been ascribed to the dazzling caused by rays of light passing through the coloboma in the iris, or to that along with some fatigue of the retina. As it is met with in other conditions than aphakia and coloboma of the iris, and is not a very common occurrence, it is impossible that these should in themselves be of paramount influence in the production of the erythropsia. When it appears after a cataract extraction, it seems usually not to come on until some time after the operation. It is met with in cases, too, where everything has gone well, and good vision has been obtained.

The influence of fatigue or hyperæsthesia of the retina in giving rise to coloured vision is undoubted. Some poisons which cause nerve fatigue occasionally also give rise to chromatopsia. Thus red and green vision has been met with by Bruce and others in cases of poisoning by bisulphide of carbon. Many authors have described a condition of yellow vision, or *xanthopsia*. in jaundice. It is certainly not a common occurrence, however, and when it does occur it is probably due to a yellow discoloration of the dioptric media, which for some reason or other is not often occasioned in jaundice.

Some forms of subjective light sensation are of cerebral origin. One of the most common is that associated with *hemicrania*. It may begin in different ways, and last for a longer or shorter time, usually after prolonged bodily or mental fatigue, or at a time when there is more or less nervous exhaustion from want of food or sleep; it suddenly makes its appearance as a dark spot to one side of the field of vision in both eyes. The dark area slowly increases in size, and after some time becomes bordered by scintillating and often coloured, zig-zag, margins of greater or less intensity. The configuration of these margins of light often resembles the angular wavy outline of a fortification, and for this reason Airy gave to the affection, from which he himself suffered, the name of *teichopsia*. The duration of the whole visual disturbance is generally less than half an hour. The appearances fade away from the centre towards the periphery, and are most frequently followed by severe headache, often accompanied by sickness, which lasts for several hours. The central nature of the subjective sensations just described is pretty definitely established by the fact that they may occur, and indeed this is most frequently the case in true hemianopic form.

This could only be the case when the temporary disturbance, whether vasomotor or of whatever other nature, was situated centrally with respect to the chiasma, unless possibly a vasomotor disturbance took place simultaneously in symmetrical halves of both retinæ. The latter possibility is unlikely, because it would not be in accordance with the anatomical arrangement, so far as is known, of the nerve fibres which supply the retinal vessels, but more especially because no such disturbance can be detected by ophthalmoscopic examination during the occurrence of the symptoms. On this latter point I am able to confirm the observations made by Förster, Haskett-Derby, and others. The occasional occurrence at the same time of other cerebral symptoms is another circumstance which is suggestive of a central origin. An interesting experiment, too, which points to the same conclusion, was made by Kums. He found that phosphenes, or subjective sensations of light produced by pressure, could not only be perceived during the attack, but that these were referred to a different plane from that to which the scintillating scotomata were projected. It seems probable, though, that when the hemianopic or bilateral character is not marked, the disturbance may in some cases really proceed from the retina. Thus I have occasionally met with eccentric negative scotomata in one eye, which appear to have been the result of attacks of this nature. It would be interesting to try the result of Kums' experiment during the attack in such cases. Another kind of central subjective coloured vision, which is probably much rarer than that connected with hemicrania, takes the form of an aura preceding an epileptic fit. This I have not seen, but it is described by Hughlings Jackson, and seems most commonly to be an erythropsia, everything appearing to the patient intensely red.

IDIOPATHIC NIGHT BLINDNESS.—If the eyes be subjected continually, day after day, to a more than usually intense light, such as is reflected from the surface of the sea or plains in tropical climates, or from chalk-pits or snow, there is apt to be set up a condition which has been called *idiopathic night blindness*. The condition arises from the illumination of the whole retina by strong irregularly reflected or scattered light; other symptoms, viz., a central scotoma due probably to coagulation or some other molecular change in the retina, are caused by the direct action of the light from strong sources of illumination, such as the sun or electric arc.

Physiologically there exists a condition in some respects allied to night blindness. Thus, if we suddenly enter a darkened room, after our eyes have been exposed to the full light of the day, we experience at first a much greater difficulty in recognising objects around us than we do after the lapse of some minutes spent in the comparative darkness. What is called an adaptation of the retina takes place gradually, and is longer in attaining its maximum the more intense has been the illumination to which the eyes were previously subjected. The time necessary for complete adaptation is also subject to individual differences, depending, amongst other conditions, on the state of the health.

Idiopathic night blindness is more especially liable to occur if, along with the exposure to strong light, the individual is the subject of some weakness,—malnutrition, anæmia, scurvy, &c. And although in much the greater number of cases of idiopathic night blindness the primary exciting cause has been found to be referable to the action of strong light alone, or combined with such conditions as those mentioned, a certain proportion of cases appear to occur as a result of these debilitating circumstances independently of any abnormal conditions of illumination. Of this nature are the cases described as occurring in women shortly before confinement, and in some cases of cirrhosis of the liver and jaundice. The few cases which I have seen of idiopathic night blindness, which is much more common in the southern countries of Europe and in the Tropics, have been in sailors and soldiers who have returned from these regions. The affection apparently begins with a condition closely resembling that just described as physiological, but in which the period occupied by the adaptation of the retina is very much prolonged. Eventually the adaptation has not time to become complete before the individual is again subjected to the unfavourable conditions, and so a true night blindness is more or less gradually acquired. The difficulties of vision come on during dusk. In rooms, too, which are illuminated by artificial light, unless the illumination is pretty powerful, only objects directly illuminated by the source of light are seen distinctly, the others being more or less indistinct, according to the severity of the symptoms.

The condition is one of anæsthesia or torpor of the retina, which requires an abnormally strong stimulus to awaken its physiological activity. Other symptoms are found at the same time depending more or less on the same cause. Thus there is often a restriction of the field of vision to an extent which varies in different cases; there is, too, a certain amount of defect in the vision for blue, which leads to a tendency to confuse between shades of blue and green, a condition which is not characteristic of any form of true congenital colour blindness. Alfred Graefe has also described as complications defective accommodation and convergence, as well as a diminished tendency towards the fusion of double images produced by prisms. These are probably due to defective stimulation to reflex or associated actions, owing to weakness of the afferent stimulus. They disappear along with the night blindness. According to Reymond, the visual acuity of individuals suffering from idiopathic night blindness does not begin to diminish until the strength of the illumination is reduced to a point which begins to tell on the normal acuity, but from this point there is a very much more rapid deterioration than in normal eyes as the illumination is further reduced.

The indication for *treatment* is to withhold light so as to allow the retina time to recover itself; and in complying with this indication it is not necessary to keep the patients in absolute darkness, but merely in considerably subdued light, either by the use of dark spectacles or residence in a darkened room, while at the same time attention should be directed, if necessary, to means calculated to improve the general health. Treatment carried out on these lines is always successful, and usually after a very short time. There appears, however, to be a decided tendency to relapse, which should be guarded against by a prolongation of the treatment after recovery.

In a considerable proportion of cases of idiopathic night blindness there is also a condition of xerosis of the conjunctiva due to the glare which gives rise to the defect of vision. A micro-organism, supposed to stand in some causal connection with this xerosis, has recently been discovered.

Idiopathic night blindness is accompanied by no marked or constant ophthalmoscopic changes. According to Poncet there is constriction and dilatation of the veins of the retina, conditions which, if they exist at all, have certainly little to do with the characteristic symptoms. This absence of ophthalmoscopic changes is also generally characteristic of the somewhat rare condition of congenital night blindness, although it is highly probable that it is closely allied to the degenerative change of the retina known as retinitis pigmentosa; possibly it may be the same disease occurring during intra-uterine life. Cases of congenital night blindness have not, however, generally been found to be complicated by restriction of the field of vision, so that they may be due to some defective development, especially as they remain stationary during life.

Experiments of Charpentier and others have demonstrated pretty conclusively that there are two distinct retinal end organs, through the medium of which the transformation of physical states into the nervous stimuli leading to vision, takes place. One of these merely effects the transformation of energy which gives rise to the perception of light, and is therefore, so to speak, the end organ for the light sense. The other is capable of differentiating the impressions which it receives (or, it may be, the difference of the impressions formed on the two end organs), so as to lead eventually to a consciousness of the varying intensities and quality of the light rays falling on different parts of the retina, and thus give rise to the sensations of form and colour. What may be called physiological night blindness shows that on exposure to light there is produced a certain degree of exhaustion of the light sense end organ, so that a certain time has to elapse before it recovers itself sufficiently to be capable of its full delicacy. As has been said, too, abnormal stimuli are capable of very much intensifying the state of exhaustion, and consequently of prolonging the period necessary for recovery. Thus it becomes a mere question of the balance between supply and demand, so that it is evident that an abnormal degree of exhaustion may be occasioned by either an excessive demand on the one hand, or a defective supply on the other. There is therefore no occasion to relegate to different categories of causation the idiopathic night blindness caused by strong light, and that resulting occasionally from debilitating disease or from altered conditions of the blood, as both may evidently be due to exhaustion in the same end organ. What that end organ probably is, we learn by a study of the more distinctly pathological conditions associated with the symptom of night blindness, for we find then that whenever there is absence or destruction of the retinal pigment cells, be it congenital or the result of inflammatory or degenerative changes, there night blindness is a more or less marked symptom, and this independently of whether other elements of the retina are affected or not.

MUSCÆ VOLITANTES.—The appearance of shadows in front of the eye may or may not be pathological, according to their nature, and the conditions under which they are seen. There are many irregularities in the normal eye which interfere to a slight extent with the passage of the light rays to the retina, and therefore cast shadows on it. These shadows are, however, mostly too faint to be perceived, owing to the small size of the bodies which throw them, compared to the extent of the surface of light from which rays pass through the pupil. The shadow thrown by one point of light is therefore illuminated by the rays proceeding from others. Only such bodies as lie very close to the retina are rendered at all visible by their shadows, and even then are, as a rule, so faint as to escape observation.

Many people notice small faint shadows which they project to different distances in front of their eyes when looking at a uniformly illuminated surface, such as a white cloud or a sheet of white paper. These have different shapes; they are annular, or strung together in beaded chains, or have more the appearance of irregular shreds of tissue. They are not as a rule fixed, so that while following the movements of the eye they are generally observed to change their position as soon as the eye is brought to rest. On looking upwards, for instance, they appear first to be thrown up along with the eye. On this account these small faint shadows are called *muscæ volitantes.* They are caused by the existence in the vitreous chamber of small portions of tissue, probably in most cases embryonic. The fact that these shreds of tissue throw shadows at all, under ordinary circumstances, shows that they must be at the posterior part of the vitreous. Owing, too, to the free movement of these muscæ, the vitreous must be more or less fluid in the portion occupied by them. As, however, the same muscæ can always be seen over and over again, pretty much at the will of the individual, and differ very little in their faintness, the fluid portion in which they float must be very narrow. When the rays which enter the eye proceed from a luminous source of a very small extent, such as is the case with those which pass through a pinhole in a card held close in front of the eye, the muscæ appear much darker and more numerous, and other more anteriorly placed irregularities become at the time visible.

It is not easy to draw the line between what may be looked upon as pathological, in respect to the appearance of muscæ volitantes, and what is merely physiological. With a small pupil and continued fixation, such as is necessary for writing or drawing on strongly illuminated sheets of white paper, &c., the muscæ seldom fail to be observed, though they are more readily seen where there is myopia, and the surface looked at lies beyond the far point. When attention is once drawn to them, they frequently cause considerable annoyance. Yet under such conditions their appearance cannot be considered other than physiological. On the other hand, when the conditions are not specially favourable, and they yet cause more or less constant annoyance, they are an indication of the existence of a hyperæsthetic state of the retina, and as such often of some general disturbance, most frequently in connection with the liver or other digestive organs. When numerous and changeable, they point to some degree of liquefaction of the posterior part of the vitreous, and such cases are often associated with the higher degrees of myopia. As long, however, as the bodies casting the shadows are so small as not to be recognisable on ophthalmoscopic examination, they may be generally diagnosed as mere muscæ. The larger floating opacities in cases of disease of the vitreous, or hæmorrhage into the vitreous, are visible ophthalmoscopically, so that the complaint of anything appearing to float in front of the eye should always lead one to make a proper objective examination.

Colour Blindness.—Congenital defects of colour vision occur in from three to four per cent. of the male population of civilised countries. Amongst females the percentage is enormously much lower, or only about one-twentieth of that for males. This comparative frequency of defects of colour vision has directed attention to the possible dangers which might result from the employment in our railways and mercantile fleet of individuals unable to distinguish with certainty between red and green, the colours universally used as signals. Accidents directly traceable to mistakes arising from colour blindness must be of extremely rare occurrence, partly because the two signal colours are not amongst those pairs for which the greatest confusion exists, and partly because the recognition of the signal does not always depend upon one man alone. The possible dangers have certainly been considerably exaggerated. Still there can be no doubt that a systematic examination of the colour vision of all persons entering these services is desirable.

When the possible dangers in connection with colour blindness were recognised, it naturally became of importance to discover a means whereby any trace of this anomaly could be speedily detected. Holmgren has the merit of being the first to devise and employ a method which has proved to be thoroughly practical and expeditious. It is a well-known fact that a high degree of achromatopsia may co-exist with a tolerably perfect power of naming colours. It is evident, therefore, that any system based on the statements made by individuals as to the names of colours presented to them must be rejected as impracticable. Holmgren, therefore, adopted the method of *comparison* between colours which to the normal eye are different; a method which, in a less perfect manner—inasmuch as the time required for examination is much longer—was used by Maxwell and Seebeck.

Holmgren's test for colour blindness.—The individual examined is asked to pick out from amongst a large number of different coloured wools those which appear most like one particular shade placed before him.

From the way in which the test is executed by different individuals, it can at once be seen whether they are colour blind or not. Those with normal vision, provided they are possessed of a certain amount of intelligence, are not long in selecting the few shades which most nearly resemble the pattern given them to match. A colour blind individual, on the other hand, soon makes a sufficient number of mistakes to reveal his defect. In order, however, at once to obtain some idea as to the nature of his colour blindness, it is advisable to choose certain colours as patterns. Holmgren begins with light green, and when mistakes are made with this, proceeds with some shade of rose—that is, a colour between red and blue. This is very well suited for this purpose, as it can at once be seen, from the colours with which it is confounded, in which direction the defect lies.

Several subsequent investigators have rejected as superfluous the preliminary examination with light green, and begin at once with the rose colour. This is a mistake, as although rose probably suffices as a test for all forms of colour blindness when complete, yet slight anomalies of colour vision are most easily detected by using light green. The reasons for choosing wool instead of coloured glass, paper, pigments, &c., are that all colours and shades of wool are easily obtained, and can be used without any preparation; that, having the same colour on all sides, any piece of one colour is easily recognised amongst a lot of others differently coloured; that the rough surface causes no difficulty by reflection; and finally, that wool is easily packed and carried about.

Holmgren gives as the result of examination by his method the following classification of all cases of defects of colour vision:—

1. *Total colour blindness.*—In these cases colour hues are not distinguished from each other as such, but only according to their relative brightness (very much in the same way as the normal eye would distinguish coloured objects illumined by a sodium flame alone).

2. *Partial colour blindness.*—This may be (*a*) complete, or (*b*) incomplete. Complete partial colour blindness he divides into (*a*) red blindness, (*b*) green blindness, (*c*) violet blindness.

Although most writers are agreed as to the great superiority of the above described method for the rapid determination of colour blindness, there is a want of unanimity with regard to the classification of cases of partial colour blindness; some—amongst whom are Hering, Cohn, Stilling, &c.—contending that there is no difference between red and green blindness, and between blue and yellow blindness, whilst others—Donders, Raehlmann, Magnus, &c.—follow Holmgren's classification, which is based on what is known as the Young-Helmholtz theory of colour perception.

What we wish to know is, what is the actual condition of the sense of colour in those who are colour blind? In the first place, there can be no doubt that an individual who is blind for one particular hue is at the same time blind for its complement. That this is the case is shown by the following facts. It is possible by the rapid rotation of a disc to obtain from three or more suitably selected coloured sectors an impression which is identical with that of a mixture of black and white produced in the same way; the colours taken in certain proportions can be got to neutralise each other, so that the resulting impression is colourless. The slightest removal of any portion of one of the colours entering into the combination can at once be detected, and some colour sensation is the result. If, on the other hand, the disc should contain two sectors of exactly complementary colours, their simultaneous removal does not destroy the colourless effect; the remaining colours continue to neutralise each other, so that the impressions they give rise to, following each other in rapid succession, resolve themselves into grey. Now it is found that the same mixture which to a normal individual appears similar to a mixture of black and white, also appears so to the colour blind individual, whence it follows, as they are known to be blind for one colour that they must either be insensitive to two complementary hues in both discs, or to only one in each. If they only fail to perceive one, both discs must appear to them coloured; but this is extremely unlikely, because then all objects which appear to the normal eye colourless must appear to them coloured. There is indeed no reason to suppose that this is the case. But there is another reason for believing that it cannot be so, viz., the analogy which exists between physiological colour blindness at the periphery of the retina and the normal colour sense on the one hand, and pathological colour blindness on the other.

Adamük and Woinow have found that the colour mixtures which appear grey at certain parts of the periphery of the retina vary according to the intensity of the illumination. According to Donders and Landolt, too, the peripheral colour impressions do not differ from the central if the intensity of illumination be increased. Although the colour blindness existing for the peripheral parts of the field of vision is apparently therefore only partial, still the fact remains that a very similar colour confusion exists for those parts of the normal field, as in the colour blind at the point of fixation. Thus a red or green object will appear when viewed peripherally yellow, grey, or blue, according to the hue taken; yet a white or grey object does not become coloured by being removed from the centre to the periphery of the field of vision, which, as a white surface reflects all rays equally, or at any rate nothing but rays which neutralise each other, would necessarily be the case if certain parts of our retina were insensitive only to particular and non-complementary homogeneous rays. Again, if a certain colour appear colourless to a colour blind individual, its after-image appears also colourless, and one which fails to produce the normal impression also gives rise to an after-image which is exactly complementary to the impression received, and not to that which would be seen by any one whose colour sense was not defective. There is no reason, however, why rays which, although colourless, are yet visible should fail to produce in us the usual successive or simultaneous contrast, unless we are at the same time insensitive to those which give rise to the complementary impression; indeed, if we have not the power of evoking the normal impression subjectively, we cannot expect to be sensitive to the ordinary objective causes which normally produce it.

Another point of importance in connection with the vision of the colour blind is, that although they fail to distinguish between many different colours, yet they are only actually blind for two particular hues which are complementary, and the slightest change in which is capable of giving rise to a colour impression. Thus most see a continuous spectrum, yet the colours seen are but shades of two hues, which are separated by a narrow band of grey. This band, too, diminishes in breadth according as the intensity of the illumination increases. These *neutral* points appear, however, to differ in different cases of colour blindness, which nevertheless are generally included under the same class. If, therefore, we could imagine all our possible colour-hue perceptions so disposed round the periphery of a circle that those which are complementary were exactly opposite each other, we should find that the directions of the diameters, representing the exact hues for which different individuals were blind, differ. Such a circle would necessarily include not only all the different homogeneous light rays which we are capable of distinguishing as different hues—in other words, all the colours of the spectrum—but also certain hues complementary to definite spectral hues, but which are not themselves found in the spectrum, which, for some reason or other, we are not able to see as homogeneous light, although we can do so subjectively, or by mixing other spectral colours.

The reason why only one neutral line is usually to be found in the spectrum of the colour blind is, that in the immense majority of cases the diameter representing the hues for which they are insensitive passes from some part of the green to some part of the space which would be occupied by the purplish hues invisible as homogeneous colours.

The position of this colour blind diameter *has an influence on the perception of all the colours of the spectrum*, and constitutes a difference—at one time small, at another time considerable—between two cases of colour blindness, so that if in one case the diameter lies between bright green and purple, the colour confusion will differ from that presented by a case where it lies between a more bluish-green and a more reddish-purple or rose colour. This, I believe, is the real explanation of the difference between green and red blindness of some authors.

Many investigators describe the spectrum of the colour blind as continuous, although only containing two colours, no portion appearing grey. This is due to the way in which they have conducted their examinations. If a very luminous spectrum be used, the images of the slit formed by the rays lying on each side of those giving rise to the exact neutral line so overlap each other as to cover the image formed by those colourless rays. A less luminous spectrum, by diminishing the colour sensations produced on each side of the neutral line, which in all cases are described as feeble, permits of the impression of a grey line separating the two colours composing the spectrum, although the line may not be very sharp. Cases do occur in which no neutral line is observed: these are cases of incomplete colour blindness, which are not infrequent, and of very different degrees. Again, if we take any red, orange, yellow, or green sector, and combine the impression received from it with that from a blue pigment by rotation on a disc, we find that the proportions of any of these with the blue, which is necessary to produce a neutral colour sensation, varies in different cases of colour blindness—that is to say, different cases are blind for different non-saturated hues, which, owing to their mixture with white light, are impure, and, owing to the absorption of light by the pigments, are wanting in brightness.

In all probability, therefore, there are a great number of forms of complete colour blindness, corresponding in general to blindness for certain rose and green hues. Although the classification into red-green blindness and blue-yellow blindness is preferable to that based on the theory of three fundamental sensations, still there can be little doubt that a more accurate one might be taken from the hues representing neutral sensations.

The term colour-blindness is in such general use that it would seem inadvisable to reject it altogether. Nevertheless, it is obvious that such a term as *colour-confusion* would be more applicable to all cases except those of total colour blindness.

The explanation of why in the case of the partially colour blind the absence of the perception of two complementary hues should leave the individual only a dichromatic spectrum, has been attempted in different ways, and has led to the various hypotheses of colour perception, none of which can be said to be more than extremely speculative. The two current hypotheses to explain the nature of colour perception are known as the Young-Helmholtz theory and Hering's theory. The reader must be referred to works on physiology or other sources for a description of these hypotheses. But, as the most important test of their value is afforded by a consideration of how far they suffice to explain the facts of colour blindness, some criticism may not be out of place here.

Both hypotheses assume the existence of so-called fundamental colour sensations. It must be admitted, I think, that the idea of fundamental colours has rather impeded than advanced our knowledge of the phenomena and nature of colour blindness. The numerous and laborious examinations that have been made with the object of detecting and classifying cases of colour blindness have been conducted mainly by those who have tried to reconcile the results obtained with one or other of the current hypotheses. The support which Young's hypothesis has received from such weighty authorities as Maxwell, Helmholtz, and Donders, etc., has necessarily given it a very firm hold of the minds of physicists and physiologists. Indeed the existence of the three different end-organs or conducting fibres which are supposed to respond in different ways to different stimuli is often referred to as little short of a certainty. Now, however satisfactory such a hypothesis may be as an explanation for colour vision in the normal state, the altered nature of the function of one or all of the end-organs, which it becomes necessary to assume in the case of colour blindness, does not adequately account for the particular conditions of colour perception which characterise that state. This, at least, is the case with reference to the colour blindness existing for the peripheral parts of the retina in everyone who has otherwise normal colour perception. This kind of colour blindness is easily studied, and the facts in connection with it, which it is of importance to consider, are as follows:—For the outermost portions of the retina there is total colour blindness. For a less peripheral zone the defect is only partial (red-green blindness). In neither case does the colour blindness appear to be complete, as it has been found that the boundaries of the area of correct colour appreciation are wider for very bright colours. In the partially colour blind area only two complementary hues—a particular yellow and a particular blue—are seen in the same manner as they are seen at the centre, all others appear yellowish (brownish) or bluish, and at the same time fainter or dirtier in colour, according to the hue and shade examined in this way. Two other hues may be found with some trouble which, within the same partially colour blind area appear uncoloured (neutral, grey), viz., a particular green, or bluish-green, and its complement, a particular rose-red or red. These four hues might, in a certain sense,—inasmuch, namely, as they always give rise to the same impression as soon as they become visible as coloured at all,—be called fundamental colours, and they correspond to those which, according to Hering's hypothesis, are the fundamental ones. Again, it is possible to find a great number of different pairs of hues which, although giving rise to altogether different colour impressions when viewed directly by the normal eye, appear similar when seen with the peripheral portions of the retina, as they both give an impression corresponding to some tint or shade of one of the only two hues which are there seen correctly.

Another important fact in connection with the physiology of vision in the normal eye is, that a white object when carried from the periphery to the centre, or *vice versa*, does not become coloured, but remains white. This is the natural result of the peripheral colour defect being always equal for hues which are complementary, and of the remaining dichromatic vision being one in which two complementary colours are equally visible.

Whilst, however, it is possible to compare the appearances which different hues present, when seen by the colour-blind portion of the retina, with that which they present on direct fixation, it is not possible for the normal eye to appreciate with certainty the impressions to which different hues give rise in colour-blind individuals. Colour blindness, as it occurs congenitally, is not exactly similar to the normal colour blindness of the peripheral portions of the retina. It is certainly, however, a very analogous condition, the differences being probably mainly, if not entirely, such as result from its greater completeness and the greater differences in the complementary hues which in each particular case appear devoid of any colour—*i.e.*, neutral or grey. Indeed, the results obtained by a comparison of the impressions produced in the two eyes in the few cases of unilateral, congenital, partial colour blindness which have been examined fully confirm this.

But it is not necessary that we should know exactly how the colour-blind see colours to test the validity of the Young-Helmholtz theory, we need only demand that it should afford a satisfactory explanation of the facts of normal colour blindness enumerated above. This, I venture to say, it cannot do. At all events, neither the complete loss of functional activity in one of the three fundamental end-organs or conducting fibres, nor the supposed abnormal diminution in the extent to which all respond to definite physical stimuli, can explain both the dichromatic spectrum and the unaltered impression produced by a white object.

The same objections cannot be urged against Hering's hypothesis. On the assumptions made by Hering an apparently satisfactory explanation of normal peripheral colour blindness can be given, and, therefore, presumably also of congenital colour blindness. The question rather which suggests itself is, Is there any good reason for assuming that there are any fundamental colour sensations at all? So far as I am aware, the first to seriously question the existence of primary or fundamental colour sensations was Krenchel. He pointed out that it is strange that, if there were such, we should be so absolutely unconscious of them. Who, for instance, taking the red of the spectrum, can say of any particular part of it, although the impression to which it gives rise is easily recognised from those produced by the hues on either side of it, that it is a pure red, a purer red than anything else, a primary, or a fundamental impression? What every one with normal colour vision can do, and what is therefore a test of normal colour vision is, to recognise a definite series in the hues of the spectrum which are sufficiently distinct to be recognised as different. Every one can place in a definite order a very large number of different hues, beginning with red, and passing through orange red, reddish orange, orange, yellow, etc., to violet. This is completely unintelligible to the colour-blind, but is readily apparent to every one possessed of normal colour vision, and yet no one can say that to every one with normal colour vision, the same hues give rise to identically the same impressions. The test for normal colour vision is simply that this unbroken series of impressions is recognised. This is all we know of the *psychical transformation* or elaboration of the physical conditions which form the basis of colour.

As to the *physical conditions*, much is of course known, and in this connection it may be worth while noting the following points:—In the first place, there is an unbroken series of ether vibrations of different velocity, which are capable of being appreciated as colours. The series, though continuous, does not embrace vibrations which have ever so great a ratio as 2 : 1. That is to say, there is nothing similar to the octave in sound vibrations. The series of sensations, too, corresponding to the vibrations of different velocity, is one in which a similar colour impression does not occur twice, but in which, nevertheless, we are conscious of an approximation at either end. The interval left can indeed be filled up by colours of which we may become conscious in other ways than by the action of homogeneous light on the retina. Why it should be that the number of possible different colour impressions should be greater for some parts of the spectrum than for others, and that impressions which we are capable of receiving in other ways should not be elicited by simple vibrations, is not clear.

Again, the same colour sensation—as far at least as hue is concerned—is got not only from the effect of vibrations of one velocity alone (homogeneous light) on the percipient elements of the retina, but also from the simultaneous action of vibrations which, acting alone, would give rise to impressions of colour occupying positions on either side of it in the series of possible colour sensations. This fact, taken along with the incompleteness of the octave of colour-eliciting vibrations, and the great difference in the nature of the vibrations themselves, is, as it appears to me, sufficient to exclude all analogy between the sensations of sound and colour, which is, nevertheless, often assumed to exist. The simultaneous action, then, of any two homogeneous light vibrations gives rise to an intermediary colour impression. It is, consequently, possible to select any number greater than two (therefore three at least) which, by suitable combinations, will give rise to all possible colours. This fact has no doubt contributed greatly to the notion of the existence of a few fundamental colours. The further the component vibrations are separated from each other in the spectrum, the less saturated is the resulting colour impression until it becomes altogether neutral, and the component vibrations are such as give rise to complementary colours. If, then, we assume, as Hering does, that there are only four fundamental colour sensations, and that all others are simply combinations of two of these, it seems not only strange that we should not be conscious of a particular purity in these fundamental colours, but that there should be such an equal sensation of saturation about all the spectral colours.

We know, then, the primary cause of colour sensations, and the final result. Of the intermediary stages in the process—viz., the manner in which the definite physical conditions become changed into definite psychical impressions—little is known. It is known, of course, that the first transformation takes place in the retina, and that the brain receives the retinal impressions, which are conveyed to it along the optic nerves. But we do not know what is the nature of the transformation that takes place in the retina, or of the resulting primary excitation in the brain which awakens the consciousness of colour. It is natural that there should be speculation as to the manner in which the intermediary process is accomplished. One can readily understand, too, that the fact that it is possible to obtain all colours from three or more colours variously combined, taken along with the doctrine of specific nerve energies of Johannes Müller, should create a strong leaning towards the fundamental colour hypotheses. Apart, however, from this, there does not appear to be any reason for making such an assumption; there is, in fact, no evidence of the existence of primary colours either in the physical basis or in the final consciousness of colours; in other words, in all we know anything about.

A question which naturally suggests itself is, Where is the defect in colour blindness? Is it in the retina, in the optic nerve, or in the brain? So far as pathological conditions go there is never any great disproportion met with between the colour and form-sense defects in any case of purely retinal disease, that is to say, disease involving only the percipient layers of the retina; the changes in colour vision to which such alterations give rise are similar to what is caused by diminished illumination; colours are seen much as they are in semi-darkness. On the other hand, colour vision appears to be disproportionately reduced, as compared with vision for form, in all cases where the conducting mechanism is implicated. This is most marked where it may be assumed that there is a general lowering of the conductibility of contiguous groups of optic nerve fibres, as in the various forms of amblyopia affecting the centres of the retina (toxic amblyopia, retrobulbar neuritis). It is in cerebral changes, however, that the greatest proportionate defects in colour vision are found—alterations in the retinal elements and optic nerves, produce a lowering of colour perception, not merely red-green blindness, though this, especially in the latter cases, is often most marked, corresponding to the less perfect development of red-green vision at some parts of the normal retina. In the case of changes localised in the brain this tolerably equal distribution, or impaired vision for all colours, is also what is most frequently met with (in the so-called hemiachromatopsia, for instance), but cases have been recorded in which a purely red-green blindness has been acquired. The experience of pathology would render it probable then that the abnormality in colour blindness is a central cerebral one and not retinal, *i.e.*, that the retina of the colour blind responds in the same manner to the ordinary physical stimuli, but that for some reason or other the psychical result is an abnormal one. This appears to me, too, to be all the more probable from the fact that, under altogether normal conditions of colour vision, the same physical stimulus does not necessarily give rise to the same colour impression. This is seen in the well-known phenomenon of simultaneous contrast.

So far as the primary transformation of light waves goes it is certainly not necessary to assume the existence of unknown elements in the retina in order to imagine a manner in which it may respond differently to qualitatively different stimuli. It has long been known that alterations in the pigment of the hexagonal cells of the retina are associated with defects in the light sense, and recent experiments by Engelmann, Van Genderen Stort, and others, have shown that changes actually take place in the disposition of the pigment in these cells by the action of light. The retinal pigment must therefore be looked upon as in some sense an end-organ. It is at least not impossible, then, that a different proportionate stimulation may be produced in the cones and pigment for each appreciable difference in wave length or combination of homogeneous stimuli. What would seem more likely, however, inasmuch as the colours resulting from homogeneous light are always more saturated than the similar ones otherwise produced is, that the stimulation of the pigment in each case merely gives rise to the sensation of light, while the different action of different wave lengths and the equivalent more complicated stimuli on the cones, in some manner elicits the qualitative differences in the impressions produced. That there is some intimate connection between the stimulations of those two end-organs in their relation to colour perception is apparent too from the fact that colour impressions are different according to the degree of illumination if below or beyond a certain intensity. The shortening of the spectrum, too, met with in some colour-blind people, and the incompleteness of the octave of colour impressions from homogeneous light may have something to do with this. The shortened spectrum has, so far as I know, not been shown to be a peculiarity of the colour-blind state.

The greatest stumbling block, however, is presented by the physiological notion of specific nerve energies. According to this doctrine it would appear unlikely that such a multitude of different impressions could be conveyed to the brain, as must necessarily be the case if there were there a direct consciousness of the differences in the manner in which the transformation of energy took place in the retina for each and every different colour which it is possible to distinguish. But we cannot say that such is not the case. Some have imagined a transmission by periodic variations in the nerve currents (which, of course, is the only energy transmitted along the nerve), leading to a reproduction of the same physical state in the cerebral cells at the visual centres, analogous to the manner in which sound vibrations are reproduced in the telephone. In the telephone the velocity of the current which transmits and reproduces the periodic vibrations is enormously rapid as compared with the vibrations. Light vibrations have, on the contrary, such a much greater velocity than the nerve current that it is almost inconceivable that there could be a similar transmission, that is to say if we imagine an altogether analogous and, so to speak, direct transmission. It is not inconceivable, however, that the transformation of energy which takes place in the retina, may be of such a nature as to render its reproduction,—that is to say the reproduction of the same molecular state in the brain,—quite compatible with the slower transmission of energy along the optic nerve. At all events, it is impossible to say that this is not or cannot be the case.

If we imagine, then, that the different way in which the different physical causes produce stimulation of the end-organs, each give rise to correspondingly different states in the molecular activity of the cerebral cells, then we might suppose further, that when colour-vision is normal the molecules may be put into their particular state of activity equally well by all the different physical stimuli capable of giving rise to different colour impressions. In the colour-blind, on the other hand, we might suppose that only one particular state (or rather two connected in some intimate manner) can be set up by all these same physical causes of stimulation. The resulting impressions in the case of the colour-blind would therefore only correspond to the normal when they were such as would in the normal brain give rise to the same impression by calling forth the same molecular state.

In a paper in the *Edinburgh Medical Journal* of 1879 I referred to the manner in which Krenchel illustrated this or an essentially similar idea. I have since been in the habit of adopting the following illustration, and I use it, of course, merely as an illustration, not as an attempt to explain what actually takes place. I use it too in preference to Krenchel's, only because I think it meets better the kind of complete independence that there seems to be in some particulars between light alone and colour. The illustration is to be found in the property that certain bodies have of polarising light. While light vibrations in all directions will pass through many transparent media, only those in one direction will pass without interruption through the polarising crystal; vibrations in planes at right angles to that particular direction are altogether excluded, and the proportion of energy transmitted as light is greater the more nearly the direction of vibrations coincides with that in which they pass without interruption, and less the more nearly it coincides with that at right angles to this. Now let us represent by way of illustration as a condition similar to that of polarised light the state of the brain cells in colour-blindness. Let us say that whereas where normal colour vision exists molecular movements or changes take place in all directions, in colour-blindness movements or changes, in one direction only, result in response to all stimuli, and that that one direction represents the two complementary hues which are properly seen.

It is evident that there must either be different molecular states set up in the visual brain cells which awaken the consciousness of different colours, or a power of analysing the different intensities of impressions in a few cells which respond always in the same manner, though with varying intensity, to the different physical stimuli, and of thus elaborating the numerous different colour sensations. The latter is what must be assumed if fundamental colours are supposed to have any existence. Now, inasmuch as both physiological experiment and pathology (*e.g.* symmetrical scotomata from brain lesions in the two eyes) point to there being a very close arrangement in the brain of the centres for corresponding points of the two retinæ, it would be natural to suppose that were the consciousness of colour aroused by the elaboration in a still higher centre of the relative stimulation of different fundamental colour brain-elements, different colours affecting simultaneously corresponding points should be blended into the proper intermediate colour-impression. But this is not the case. This fact is, as it seems to me, one argument against Hering's hypothesis. Other objections which may be advanced are, that it is by no means always the same hues which to the colour-blind are altogether colourless; that there is no consciousness of fundamental colours; and lastly, that the assumption is unnecessary, because although it may offer an explanation for the principal phenomena of colour-blindness, that condition may be explained by making assumptions which, in the present state of our knowledge, are quite as legitimate.

HEMIANOPIA.—Symmetrical defects in the fields of vision are most commonly caused by lesions in the optic nerves, the optic tracts, or the chiasma. If the defects lie to the same side in both eyes—that is, inwards in one and outwards in the other—the condition is that which is now generally called *homonymous hemianopia.* The hemianopia may be to the right or left, and partial or complete, just as we have complete and partial, right or left, hemiplegia. It may or may not be associated with hemiplegia. I have more often seen it alone, but it is probable that those in whom the visual paralysis forms but a part of the whole lesion will be more frequently met with in general hospitals. Of thirty cases observed by Schweigger, hemiplegia was absent in sixteen and present in thirteen, though in most of these it was only slight and transitory. If the case be recent and uncomplicated, there is no appearance of atrophy of the nerves. The line of demarcation between the blind and seeing portions of the field is usually sharp and regular; when the hemianopia is complete, it is a line coinciding (for peripheral portions of the field, at any rate) nearly, if not exactly, with the vertical through the point of fixation. Whether or not it also passes vertically through this point, indicating an interruption in the functional activity of half the macula lutea, is still a disputed point. My own examinations of cases of homonymous hemianopia lead me to believe that in some cases it does so, and in others not. I have most frequently found the immediate neighbourhood of the point of fixation (from 2° to 5°) apparently not implicated; but this may possibly be owing to the difficulty of securing accurate fixation. The lesion producing homonymous hemianopia has been found to occupy different situations, viz., the optic tract, basal ganglia, fibres of Gratiolet, and the cortex of the brain in the occipital lobe.

The experiments of Munk, and the results of post-mortem examination, have definitely established the existence of an unilateral source of innervation for corresponding halves of both retinæ. In the cases where the lesion has been cortical, it has either been one occupying a portion of the grey matter of the occipital lobe, or pressing on the white matter with which it is in connection, so that there is little doubt, now that Ferrier's experiments, which led him to localise the visual centres in the angular gyri, have given rise to the hemianopic symptoms, owing to a wounding of the fibres of Gratiolet.

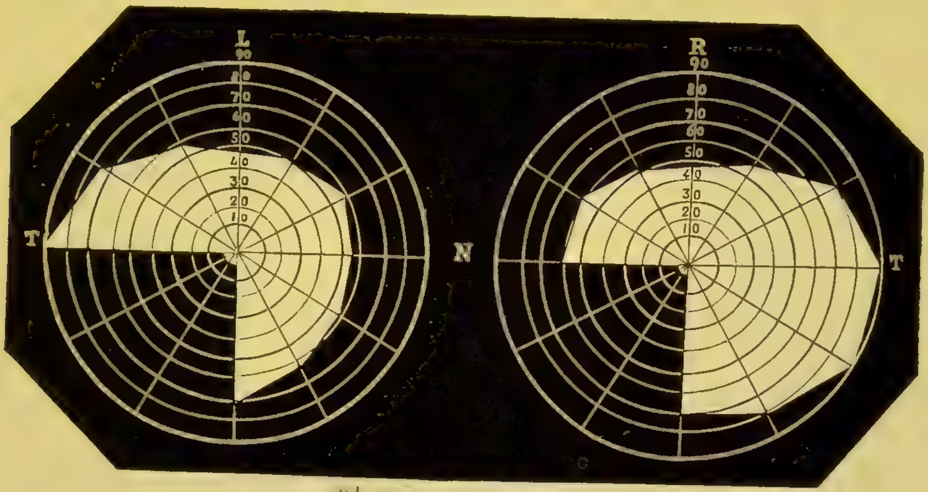

FIG. 108.—Fields of vision from a case of homonymous hemianopia, in which vision was lost in the left lower quadrant of each field.

Important data in connection with the localising of lesions which have led to hemianopia are afforded by the consideration of the concomitant symptoms, as well as by the extent and configuration of the blind area. An attempt has recently been made by Wilbrand to make use of the data afforded by the thorough subjective examination of cases of hemianopia, in order to arrive at a refinement of the diagnosis of the seat of the lesion. Wilbrand's work, though very suggestive, is necessarily to a considerable extent theoretical. He keeps constantly before him, however, the results of post-mortem examinations which he has collected, and the views he expresses are not inconsistent with the known pathology of the subject. An analysis of the subjective symptoms of hemianopia cases shows that—(1.) When in any homonymous portions of the fields of vision there is a defect in the light sense, the defects in the senses of form and colour are at least quite as extensive: (2.) When the light sense remains intact, but a homonymous defect exists for form, this is always accompanied by at least as extensive a defect for colours. Wilbrand therefore concludes that the nerve fibres connected with the centres for the senses of colour and form pass through the light sense centre. As, too, there are cases on record where the colour sense alone in the halves, or in some particular homonymous sectors of the fields of vision, had disappeared from similar portions, leaving, however, the light sense intact in these portions, Wilbrand draws the further conclusion that the centre for form must lie between the centres for colour and light, while that for colour must at the same time occupy the most central position in the brain. Wilbrand has collected many examples of actual cases of all the combinations which are possible on the above assumptions. Further investigations are called for before his views can be considered as more than speculative. In all the cases which I have seen of so-called hemiachromatopsia,—that is, cases in which the half-blindness appears to be only for colours,—I have been able by testing with very small white objects at a distance, according to Bjerrum's method (see p. 14), to demonstrate that the form-sense has also been defective over the same area. Indeed, the clinical evidences so far point altogether more to the probability of colour vision being due to a subtle differentiation of light impressions, than to any impression made upon other centres than those in which the consciousness of form are evoked.

When the hemianopia is partial, the defect is generally, though not always, of equal extent in both eyes.

Hemianopia remains as a rule stationary, even although the subjective symptoms become, in many cases after some time, associated with more or less visible changes in the discs. According to Mauthner, the most marked atrophic discoloration is found in the eye of the side to which the field is restricted. This point requires, however, further confirmation. A very unusual form of hemianopia is described by Förster, in which the defect is limited to corresponding insular portions in the homonymous halves of the two visual fields.

Two other forms of hemianopia occur, viz., temporal and nasal hemianopia. In the former the outer, and in the latter the inner half of each field of vision is deficient. They are both much rarer than the homonymous form.

The arrangement of the optic nerve fibres renders the localisation of the lesion producing temporal hemianopia a very simple process. There is, of course, the remote possibility of a symmetrical lesion in the two halves of the brain, but apart from this, and as by far the most probable cause of the symptoms, we must admit an interruption of function produced by some destruction or compression of the nerve elements of a portion of the chiasma. The defects produced in the function of the nasal halves of each retina will be more complete and symmetrical the more the lesion is confined in its effect to an antero-posterior line through the chiasma. Thus, whereas the lesion producing the more common form of homonymous hemianopia may be anywhere along the course of the optic nerve fibres between the chiasma and the cortex of the occipital lobe, and can rarely be localised with any great certainty—although concomitant symptoms often afford a clue—temporal hemianopia is almost certainly due to a lesion mainly implicating the chiasma.

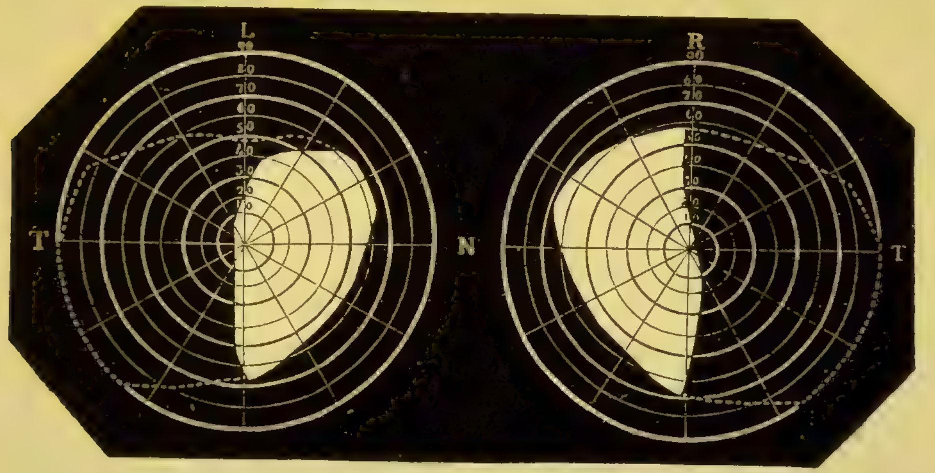

FIG. 109.—Fields of vision from a case of temporal hemianopia.

It is difficult to form any idea of the frequency of this variety of hemianopia. Owing to the nature of the defect, leaving as it does a much larger field for both eyes than the homonymous form, the patient does not as a rule complain of blindness to one side. The other symptoms, too—amblyopia, ophthalmoscopic changes, &c.—are sufficiently marked to engage the whole attention of any one who does not make a practice of examining the peripheral vision. Besides, comparatively few, no doubt, of the cases which have been observed have been published. Mauthner believes that they constitute about one per cent. of all cases of hemianopia, while Förster's estimate is twenty-three per cent.; the actual percentage probably lies between these extremes, and considerably nearer the former than the latter. It is obviously a disease of too rare occurrence to furnish even approximately correct statistics from the practice of any single observer, however extensive his experience.

A good many cases have come under my own observation, and in three of them the diagnosis—in two cases an aneurism, and in the remaining one a tumour pressing on the chiasma—was confirmed on post-mortem examination. The line of demarcation between the blind or defective portion of the field and the normal one is rarely as sharp or as vertical as in the homonymous form. The prognosis is not so good either, most cases going on to complete blindness. Some cases may remain stationary, or recover to some extent. A sudden onset, with subsequent partial recovery, or, as sometimes happens, blindness of one or both eyes followed by temporal hemianopia, is suggestive of an apoplexy in the neighbourhood of the chiasma. A slight degree of intermittence in the symptoms—slight improvement on certain days—is probably indicative of aneurism. Repeated attacks of temporal blindness, lasting several days at a time, and then recovered from, point again to acute intracranial pressure from some cause or other.

Nasal hemianopia is of very rare occurrence. The prognosis in such cases, which must depend on symmetrical lesions of the optic tracts, is extremely bad. Most, if not all, lead eventually to complete optic atrophy.

SECTION II.

CHAPTER XV.

ERRORS OF REFRACTION AND ACCOMMODATION.

THE transparent media of the eye, viz., the cornea, aqueous humour, crystalline lens, and vitreous body, are all denser than air. They offer a greater impediment to the transmission of those minute and extraordinary rapid vibrations of the ether, which, when they impinge on the living retina, give rise, through the medium of its nervous connection with certain centres in the brain, to the sensation of light.

Although light is but the consciousness of a peculiar disturbance or physical state of matter, it is convenient to use the word *light* for that state which gives rise to it.

Light, then, travels more slowly when it enters the eye than it does in the surrounding air, the retardation being different in amount in the different media, and greatest within the central layers of the crystalline lens.

Now, unless the surface of a denser transparent medium is at right angles to the direction of propagation of the wave surface of light, there is not only a retardation, but a bending or *refraction* of its direction as well.

That this is a necessary consequence of its retardation will be evident from Fig. 110, which represents the section of the surface of a wave of light emanating from a far distant point. A small portion of the surface of propagation, or the wave surface, at a great distance from the point of emanation, is approximately a plane. In the figure the plane wave surface of light meets the denser medium (II.) obliquely, so that whilst the light at A meets the surface at A_1, that at B does not reach it till B_3. By the time B reaches the denser medium, A has passed on to A_3, but as the transmission of the disturbance from A is slower in medium II. than medium I., the distance from A to A_3 is less than from B to B_3. By the time, therefore, that B has reached the denser medium it has pulled up somewhat upon A, whilst the intermediate points have all in turn received an equal check to their rate of propagation, and the direction of the plane wave surface, instead of being at right angles to AB or A_1B_1, is now at right angles to A_3B_3, and A_4B_4, *i.e.*, the direction of the propagation of light has been changed through a definite angle.

For a full explanation of how the interference of portions of the small waves of disturbance, passing from every point of the resulting wave surface, causes a change of direction of the surface as soon as the denser medium is entered, the reader must refer to works on physical optics.

Instead of considering the change in direction of the wave surface of light, we may merely consider that of the lines at right angles to that surface or the *rays of light*. When the surface is plane, *i.e.*, when the point from which the light diverges is infinitely distant, these rays are parallel to each other.

Absolute parallelism, in a mathematical sense, could only occur when the point from which the rays emanated was at an infinite distance. Rays, however, which, emanating from a very distant point,

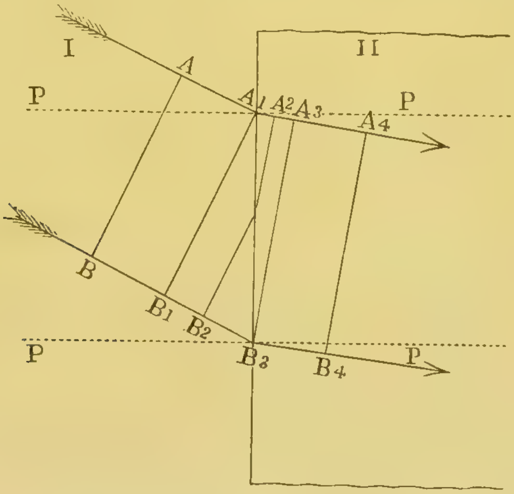

Fig. 110.

meet any surface, of inconsiderable size when compared with that distance, deviate from each other so slightly, that they may for all possible practical purposes be considered parallel.

In Fig. 110, $A\,A_1$, $B\,B_1$ represent two parallel rays, and it is seen that on entering the denser medium they are refracted towards the perpendicular (P) to the surface of that medium. Therefore rays passing from a rarer to a denser transparent medium are refracted towards the perpendicular to the surface of the denser medium, at the points where they meet that medium. The length, too, which is travelled in a definite time by the rays in the second medium, when that is homogeneous, bears always a constant proportion to that travelled in the same time in the first medium, so that $A_1\,A_2 : B_1\,B_2 = A_2A_3 : B_2B_3$, and this proportion evidently determines the degree of the separation, and must remain the same whatever be the obliquity with which the ray meets the refracting surface. This constant proportion for any homogeneous medium with respect to air is called the *index of refraction*, and is denoted by μ, *i.e.*, $\frac{B_1\ B_3}{A_1\ A_3} = \mu$. In passing from air into a denser medium μ is evidently greater than 1, from a denser medium into air less than 1.

This same proportion μ has been found by experiment in another way. Thus in Fig. 111 are represented two rays AO and CO traversing medium I., and meeting the surface of medium II. at different degrees of obliquity; i and i' representing the two *angles of incidence*, and r and r' the two corresponding *angles of refraction*, or the angles which the incident and refracted rays make with the perpendicular to medium II. at the point O.

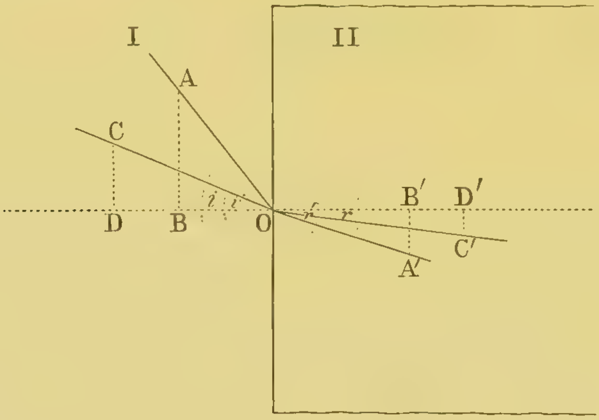

Fig. 111.

It is found by experiment that,

$$\frac{AB}{AO} : \frac{A'B'}{A'O} = \frac{CD}{CO} : \frac{C'D'}{C'O}$$

i.e., $\sin i' : \sin r' = \sin i : \sin r = \mu$ (when medium I. is air), *i.e.*, *the sine of the angle of incidence bears a constant proportion* μ *to the sine of the angle of refraction.*

The media of the eye are, however, not only refractive media, but are bounded by curved surfaces, which are approximately spherical. Owing to this circumstance they possess the property of *focussing* rays which pass into them, or, in other words, of collecting them towards a point. This is a consequence of the law of refraction just mentioned, as in the case of curved surfaces the perpendicular to the surface at any point coincides with that of the radius at that point. In Fig. 112 is shown the denser medium bounded by a spherical surface, and the parallel rays which meet it bent towards a point which is called their *focus.* Strictly speaking, only monochromatic rays which suffer refraction at points equidistant from the *axis*, or line joining the centre of the spherical surface with the point from which the rays proceed, are focussed at the same point.

The distance separating the foci of different hollow cylinders or cones of light, formed by rays meeting the spherical surface at points equidistant on all sides from this axis, differs not only with the degree of their separation, but is also greater for equal amounts of separation, the farther the component rays are removed from the axis. This gives rise to what is called *spherical aberration.* In speaking of the focus of any point of light, therefore, what is meant, as a rule, is that point to which the rays *tend* to become gathered, the more nearly they coincide with the axis.

If the point from which the rays emanate be at an infinite distance, so that they meet the refracting surface parallel to each other, the focus of the axial rays is called the *principal focus* (Fig. 112 *f*).

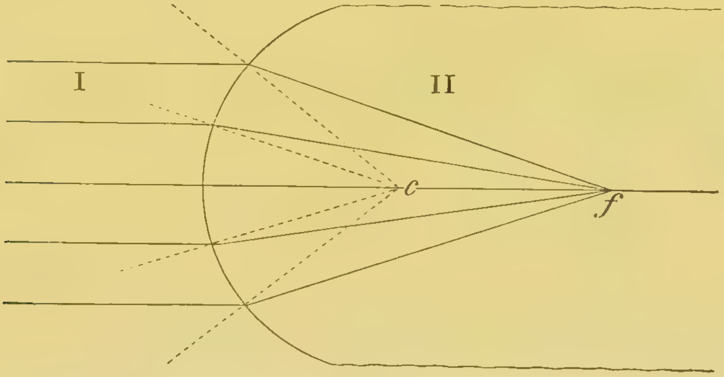

FIG. 112.—Shows the deflection of rays towards a focus, *f*, when the denser medium is bounded by a curved surface whose centre is at *c*.

Practically, in order to obtain an approximate focus, or, in other words, to get rid of spherical aberration, it is necessary to exclude all but the more axial rays, *i.e.*, the aperture of the spherical refracting surface must be small compared to its circumference as well as compared to the distance from it from which the rays proceed.

In the eye spherical aberration is partially corrected by the surfaces of the refracting media being not exactly spherical, and by the manner in which the different more or less centred refracting surfaces are relatively disposed. The remaining aberration, which must be pretty considerable, seems to be of little consequence, mainly probably on account of the arrangement of the percipient elements of the retina as discrete and separately stimulable end organs.

If we know the radius of curvature and the index of refraction of any refracting medium, as well as the distance of a point of light or object from it, it is easy to find its focus or image. This may be done by at once assuming that the incident and refracted rays make such small angles with the perpendicular, that the values of the angles may be substituted for those of their sines. But it is better to get an expression for the general reciprocal values of the distances of object and image along the axis for every angle of incidence and its corresponding angle of refraction, and then to deduce the particular values, on the assumption that the aperture is so small, that it may be neglected, compared with the radius of curvature and the distance of the object. These values are then called the *conjugate focal distances*. In Fig. 113 we have, putting u for the distance of the object and v for the distance of the image along the axis—

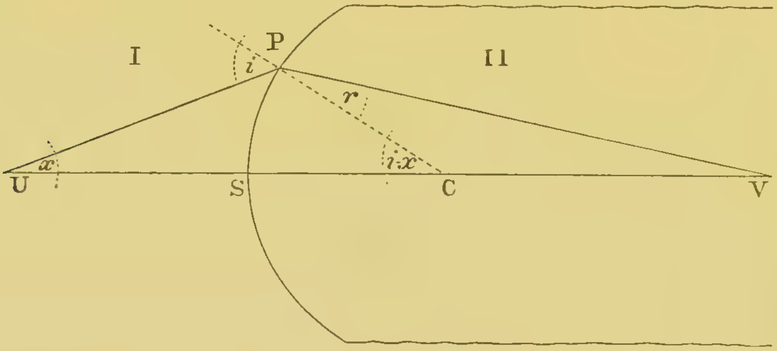

Fig. 113.

$$u = \mathrm{U\,S},\; v = \mathrm{S\,V} = \mathrm{S\,C} + \mathrm{C\,V}$$
$$= \mathrm{R} + \mathrm{C\,V}$$

and C V : R = sin C P V : sin C V P = sin r : sin $(i - r - x)$ where $i = \angle$ of incidence, $r = \angle$ of refraction, and $x = \angle$ between the two rays which diverge from v.

$$\therefore\; \mathrm{C\,V} = \mathrm{R}\,\frac{\sin r}{\sin\,(i - r - x)}$$

$$\therefore\; v = \mathrm{R}\left(1 + \frac{\sin r}{\sin\,(i - r - x)}\right)$$

On expanding $\sin(i - r - x)$, and remembering that $\sin i = \mu \sin r$, the value of v becomes

$$v = \mathrm{R}\left\{1 + \frac{1}{\cos x\,(\mu \cos r - \cos i) - \sin x\,(\cos i \cos r + \sin i)}\right\}$$

and, finally, putting for $\sin x$ its value $\mathrm{R}\,\dfrac{\sin i}{\mathrm{U}+\mathrm{R}}$

$$v = \mathrm{R}\left\{1 + \frac{1}{\cos\sin^{-1}\dfrac{\mathrm{R}\sin i}{u+\mathrm{R}}\,(\mu\cos r - \cos i) - \mathrm{R}\dfrac{\mu\cos r\cos i + \sin^2 i}{u+\mathrm{R}}}\right\} \quad 1)$$

When u is infinite, *i.e.*, when the rays meet the surface parallel to each other, $x = o$ and the expression for v reduces to—

$$v = \mathrm{R}\left(1 + \frac{1}{\mu \cos r - \cos i}\right) \quad . \; . \; . \; . \; . \; . \; . \; . \; . \; . \quad 2)$$

The conjugate focus for u is got by putting i and $r = o$, that is, the limit to which the angles approximate, as the rays more and more coincide with the axial one, 1) then becomes—

$$v = \frac{\mu\, u\, \mathrm{R}}{u\,(\mu - 1) - \mathrm{R}} \quad . \; . \; . \; . \; . \; . \quad 1\,a.)$$

and 2) becomes

$$v = \frac{\mu\,\mathrm{R}}{\mu - 1} \quad . \; . \; . \; . \; . \; . \; . \; . \quad 2\,a.)$$

1 *a*) is usually written—

$$\frac{1}{u} + \frac{\mu}{v} = \frac{\mu - 1}{\mathrm{R}} \quad . \; . \; . \; . \; . \; . \; . \quad 1\,b.)$$

The longitudinal spherical aberration, or the distance along the axis separating the foci of the axial and any other ray, is got, for diverging and parallel rays, by subtracting respectively 1) from 1 *a*, and 2) from 2 *a*.

When v is put $= \infty$, in 1 *b*), the value of u becomes $\dfrac{\mathrm{R}}{\mu - 1}$, while that of v, when u is put $= \infty$, is $\dfrac{\mu\,\mathrm{R}}{\mu - 1.}$ If we denote these values of u and v respectively by f and f', the *principal foci*, we readily arrive at a formula which connects the principal foci with any other pair of conjugate foci; for, noticing that

$$f' = \mu f$$

$$i.e., \quad \mu = \frac{f'}{f}$$

1 *b*) may be written

$$\frac{1}{u} + \frac{f'}{f} \cdot \frac{1}{v} = \frac{1}{f}$$

and multiplying by f—

$$\frac{f}{u} + \frac{f'}{v} = 1. \quad . \quad . \quad . \quad . \qquad (3)$$

This is one of the most important formulæ in geometrical optics, so far as the eye is concerned, and we shall see that it holds good for any number of centered refracting surfaces as well, provided these quantities, f, f', u, and v, be measured from certain points to be afterwards considered.

Besides spherical aberration, the eye is not free from chromatic aberration—that is to say, that the different rays of the spectrum which are contained in ordinary daylight are not all refracted to the same extent, so that the focus for the blue rays, for instance, is shorter than that for the red.

In considering the eye at rest, and not exerting the power which it has of altering its state of refraction, or, in other words, of *accommodating* for the distance from which rays proceed, many points may be perfectly well explained by looking upon it as consisting of only one dense transparent medium, bounded by only one surface, instead of, as is actually the case, being a very complex instrument, with a number of differently refracting media.

When the eye is in a state of rest, and not exerting any of its power of accommodation, *i.e.*, when its refracting power is at its lowest, one of three conditions must be represented in every case:—(1.) the principal focus (or the focus of rays which meet the eye parallel to each other, and therefore proceed from an infinitely distant point) coincides exactly with the most sensitive portion of the centre of the retina; (2.) the principal focus lies in front of the most sensitive portion of the centre of the retina; and (3.) the principal focus lies behind the most sensitive point of the centre of the retina. In the first case, the eye is said to be *emmetropic*, in the second *myopic*, and in the third *hypermetropic;* and these conditions of refraction are called respectively *emmetropia*, *myopia*, and *hypermetropia.*

Emmetropia in the true sense is exceedingly rare. Slight deviations in the direction of either myopia or hypermetropia are common, however; so that the eyes which are *practically* emmetropic are common.

It is evident that as there are three elements on which the state of refraction depends—viz., the length of the antero-posterior axis of the eye, the curvature of the refracting or dioptric media of the eye, and the refracting power of these media—the conditions on which myopia or hypermetropia depend might be abnormal conditions of all or any of these elements. As a matter of fact many cases of myopia and hypermetropia do occur which are wholly or mainly caused by some abnormality in each of these directions. Thus we have *axial* myopia or hypermetropia, due to a too long or too short antero-posterior axis of the eye (Fig. 114); *curvature* myopia or hypermetropia, caused by too great or too little curvature (Fig. 115); and *index* myopia or hypermetropia, as the result of too great or too feeble refracting power.

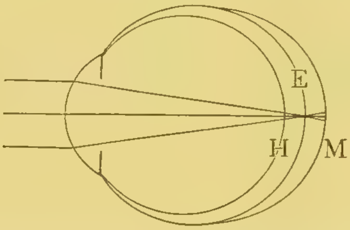

FIG. 114 shows the complete focussing of parallel rays in the case of emmetropia, E, and incomplete focussing in hypermetropia, H, and myopia, M.

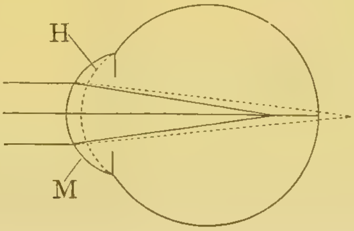

FIG. 115.—Showing the stronger refracting power of the more highly curved cornea (continuous line).

The emmetropic eye alone when at rest is focussed for parallel rays. Rays diverging from a point in front of the eye and suffering the same amount of refraction would meet behind the retina, whilst rays converging to a point behind the eye, and also refracted to the same extent, would meet in front of the retina. This is represented in Fig. 116, in which the conjugate focus of rays diverging from A is represented at A′ behind the retina, while the conjugate focus of rays converging towards B is represented at B′ in front of the retina.

It will be evident now that a myopic eye (*i.e.*, one in which the retina lies behind the principal focus of the refracting media) is, in a state of rest, focussed only for rays diverging from some point at a particular finite distance in front of it, and also that a hypermetropic eye (*i.e.*, one in which the retina lies in front of the principal focus of the refracting media) is in a state of rest focussed only for rays which converge towards a particular point lying at a finite distance behind it.

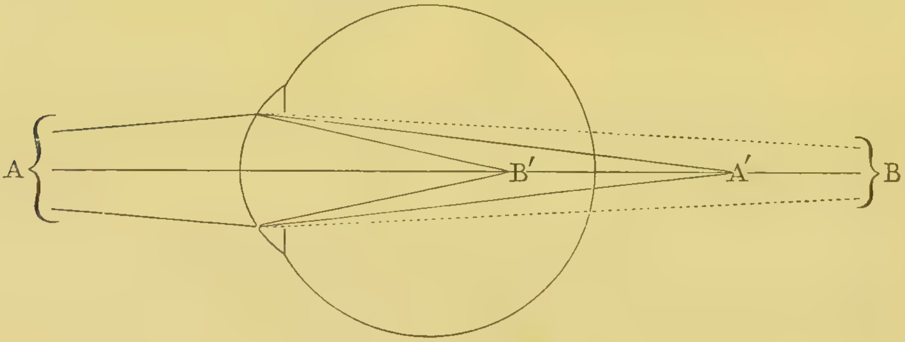

FIG. 116.

Therefore the *static refraction in emmetropia leads to the focussing of parallel rays on the retina, the static refraction in myopia to the focussing of diverging rays on the retina, and the static refraction in hypermetropia to the focussing of converging rays on the retina.*

The greater the divergence or convergence of the rays which is required in any case in order that they may be focussed on the retina the greater is evidently the error of refraction. The measurement of that error, or what is called the degree of *ametropia,* is given by the distance from the anterior part of the eye from which the rays focussed on the retina diverge, or towards which they converge. The smaller this distance is the greater is the ametropia. This distance is also called the distance to the *far point* of the eye. The far point is therefore that point from which rays which meet on the retina of the eye in a state of rest diverge, or to which they converge.

The far point in myopia lies consequently at a finite distance in front of the eye, whilst in hypermetropia it lies at a finite distance behind the eye. The far point in myopia is also said to be *positive*, in hypermetropia *negative*. In the case of emmetropia the far point lies at an infinite distance from the eye, because only then are the rays which proceed from a point truly parallel.

Practically, emmetropia is considered to exist in any case where the far point lies not nearer than 6 metres in front of or behind the eye. In the condition of static refraction, then, an emmetropic eye obtains a clear image of objects at a distance. A myopic eye in the same condition obtains a clear image of objects which lie at the distance of its far point. To a hypermetropic eye at rest, however, no objects, near or distant, are seen distinctly, as rays from every real object must diverge, and the hypermetropic eye is only focussed for converging rays.

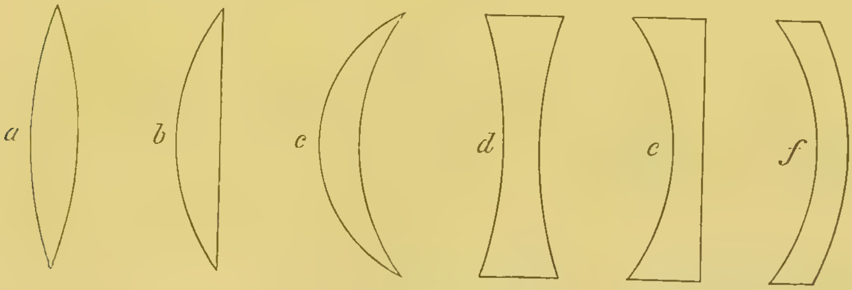

FIG. 117.—Different forms of convex and concave spherical lenses.

Spherical Lenses.—Before proceeding any further, we must consider the action of spherical lenses, that is to say, portions of highly refracting transparent substances bounded by two spherical surfaces whose centres lie on the same line which is called the *axis* of the lens, or by one spherical and one plane surface. The substances of which lenses used for most optical purposes are made are glass and crystal.

Certain names are given to lenses, according to the shape of their two surfaces. When both are convex the lens is called *biconvex.* When both are are concave, it is said to be *biconcave.* When one surface is plane, the lens is either *plano-convex* or *plano-concave,* according as the remaining surface is convex or concave. Two other forms of spherical lenses are made, in which the one surface is convex and the other concave, but with different radii of curvature. When the convex surface has the greatest curvature, *i.e.*, the smallest radius, the lens is a *convex meniscus* (Fig. 117, *c*), when the concave surface has the greatest curvature or least radius the lens is called a *concave meniscus* (Fig. 117, *f*).

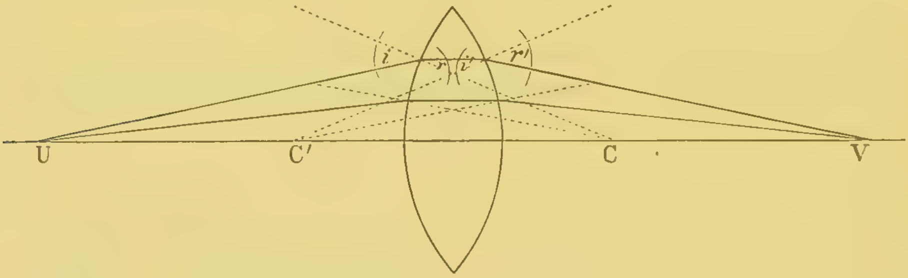

Fig. 118.—Showing the action of a biconvex lens in bringing rays to a focus.

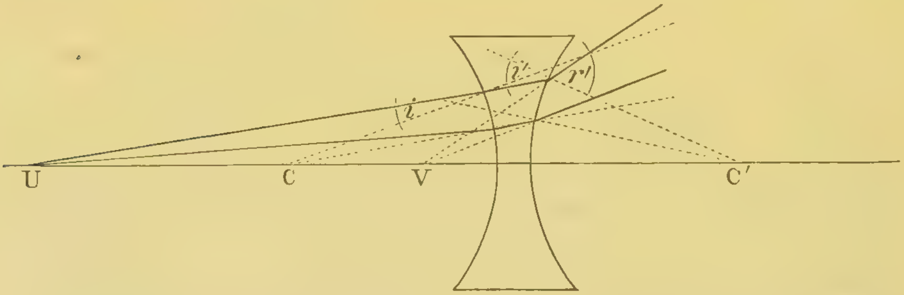

Fig. 119.—Showing the action of a biconcave lens in dispersing incident rays.

If we draw radii to the two surfaces at the point where rays proceeding from any point on the axis meet these surfaces, we may easily see how the lenses must act in refracting the rays. In Figs. 118 and 119, C and C′ are the centres respectively of the two surfaces of a biconvex and biconcave lens. The rays in passing into the lens are bent towards the perpendicular to the first surface, whilst in passing out of it, as they then pass from a denser to a rarer medium, they are bent away from the perpendicular to the second surface. This causes, in the case of a biconvex lens, a double bending towards the axis, on some point of which, and on the other side of the lens, rays proceeding from any point of the axis tend to become focussed. In the case of a biconcave lens there is produced in a similar manner a double bending of rays away from the axis, and they are thus given a divergence as if they proceeded from some point on the axis, on the same side as, but nearer to the lens than the luminous point from which these rays actually proceed. The convex lens therefore *collects* rays, whereas the concave lens *disperses* them.

Just as in the case, too, of a single refracting surface, so in the case of a lens, the focus of parallel rays is called the *principal focus,* whilst any other two points which stand in the relation of object and image to each other are called *conjugate foci.*

The principal focus in the case of a convex lens lies on the other side of it from that from which the rays proceed, and is *real*—that is to say, the rays actually are collected; and although, owing to spherical aberration, they do not meet in a mathematical point, still the more axial ones are practically focussed. Convex lenses are therefore called *positive* or *plus* lenses.

The principal focus, again, of a concave lens lies on the same side of the lens as that from which the rays proceed, and is *virtual*—that is to say, the rays do not actually, but only apparently, proceed from the focal point. Concave lenses are therefore called *negative* or *minus* lenses.

The relation between object and image in the case of a lens is easily got if we adhere to the restriction, which very much simplifies the calculation, of supposing the rays to be sufficiently nearly axial to obviate spherical aberration.

Proceeding in the same direct manner to find the focus for any ray passing through a lens, as was done for one refracting surface, we arrive at a formula which is very long and unwieldy, but which easily reduces under the limiting conditions which are postulated. It is better to consider the distance of the image formed by the first refraction of a point on the axis, the rays from which are nearly axial, as the object distance with respect to the second refracting surface, and apply the formula 1*b*. In doing so we make the assumption that the rays from a point, after refraction into the first medium, proceed towards, or appear to proceed from, another point, to which, after refraction into the second medium, a corresponding third point must exist, which will be the image of the first point. Writing 1*b* with a dash to each letter thus—

$$\frac{1}{u'}+\frac{\mu'}{v'}=\frac{\mu'-1}{R'}$$

we denote the relation existing between object and image distance with respect to the second surface, *i.e.*, for rays passing from the lens into air. In this case μ' is evidently equal to $\frac{1}{\mu}$ and $u'=t-v$, or if we neglect the thickness of the lens (t), $u'=v$. Putting in these values the formula becomes—

$$\frac{1}{t-v}+\frac{\frac{1}{\mu}}{v'}=\frac{1-1}{R}$$

and multiplying by μ—

$$\frac{\mu}{t-v}+\frac{1}{v'}=\frac{1-\mu}{R'}$$

neglecting t, and putting for $\frac{\mu}{v}$ its value from 1*b* we get

$$\frac{1}{u}+\frac{1}{v'}=\frac{\mu-1}{R}+\frac{1-\mu}{R'}=(\mu-1)\left[\frac{1}{R}-\frac{1}{R'}\right]$$

Then, writing instead of v', v which now represents the distance of the image from the lens :—

$$\frac{1}{u}+\frac{1}{v}(=\mu-1)\left[\frac{1}{R}-\frac{1}{R'}\right] \quad . \quad . \quad . \quad 4.)$$

This equation represents the relation which exists between object and image distance, for refraction through any lens. R is reckoned positive when the direction from surface to centre is the same as that of the incident light.

If u be taken infinite we get for the reciprocal value of f' the principal focus—

$$\frac{1}{f'}=(\mu-1)\left[\frac{1}{R}-\frac{1}{R'}\right] \quad . \quad . \quad . \quad 4a.)$$

$$\therefore \frac{1}{f'}=\frac{1}{u}+\frac{1}{v} \quad . \quad . \quad . \quad . \quad 4b.)$$

By making v infinite we get the same value for $\frac{1}{f}$. Therefore, when the thickness of the lens is neglected we have $f'=f$.

The sign of $\frac{1}{f}$, which determines, as we have seen, the character of the lens, is given by the signs and relative values of R and R′. In the ordinary biconvex lens, with equal curvature of both surfaces, R = R′, and they are of different signs : ∴ $\frac{1}{f}$ becomes—

$$2\,\frac{(\mu-1)}{R} \text{ and } f = \tfrac{1}{2}\,\frac{R}{(\mu-1)}$$

Further, as for ordinary crown glass, μ is approximately 1·5 (1·534 according to Brewster), $f = R$ approximately,—that is, *the focus of a common glass lens is equal to the radius of curvature of its surfaces.*

From 4*b*) it is easily seen that as $f = f'$, formula 3) also holds good in the case of a lens when the thickness is neglected, because multiplying by f we get—

$$\frac{f}{u} + \frac{f'}{v} = 1 \quad . \quad . \quad . \quad 3.)$$

It is sometimes of use in problems connected with the eye to consider the relation existing between the distance of the object from the anterior focus $(u-f)$ and that of the image from the second focus $(v-f')$:—

From 3 we have :

$$\left.\begin{aligned}
u &= \frac{fv}{v-f'}.\\
\therefore\ u-f &= \frac{fv}{v-f'} - f.\\
&= \frac{ff'}{v-f'} \quad . \quad . \quad (\alpha.)\\
&\text{Similarly :}\\
v-f' &= \frac{ff'}{u-f}. \quad . \quad (\beta\cdot)\\
&\text{Also from } \alpha \text{ and } \beta \text{ :}\\
(u-f)\ (v-f') &= ff' \quad (\gamma.)
\end{aligned}\right\} \quad . \quad . \quad . \quad . \quad 5.)$$

Use of spherical lenses in ametropia.—We have seen that in myopia the eye is too powerfully refracting relatively to the position of the retina, *i.e.*, from some causes or other its collecting power is too great. This may be rectified by putting in front of it a concave or negative lens, the strength of which is just sufficient to counteract that portion of the collective power of the refractive media of the eye at rest, which is in excess of what is required to focus rays coming from distant points on the retina. On the other hand, in hypermetropia the collecting power of the refracting media of the eye is insufficient, and this again may be corrected by putting in front of the eye at rest a convex or positive lens, the strength of which is just sufficient to supplement the defective amount.

The concave lens which *corrects* the myopia in any particular case, or the convex glass which corrects any particular case of hypermetropia, is that one which, placed at a convenient distance in front of the eye, produces in the state of static refraction of the eye the most accurate focussing of rays from a distant point (parallel rays) on the retina.

The glass in each case must be one whose focal distance equals its distance (measured from its position in front of the eye) from the far point of the eye. It must therefore have a shorter focus the nearer the far point lies to the eye. In other words, the higher the ametropia, the stronger must be the correcting lens.

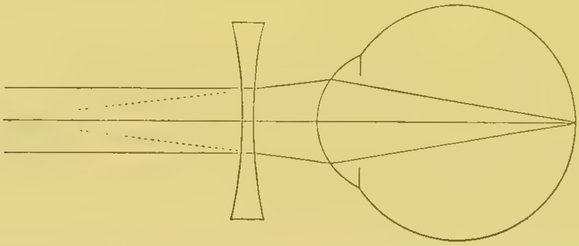

FIG. 120.—Action of a concave lens in front of the eye, causing parallel rays to enter the eye as if proceeding from a finite point in front of it.

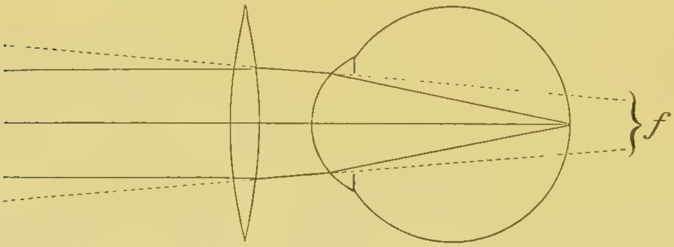

FIG. 121.—Showing action of a convex lens placed in front of the eye, causing parallel rays to enter the eye, as if directed to a point at a finite distance behind the eye.

It also follows from the above definition of the correcting glass that the nearer it lies to the far point of the eye the stronger it must be.

In myopia the far point lies at a finite distance in front of the eye, therefore the strength of a concave glass in order to correct any definite degree of myopia must be greater the further it is placed from the eye. From this it follows *that the effect of a concave glass of definite strength on myopia is weakened by removing it from, and strengthened by approximating it to, the eye.*

In hypermetropia the far point lies at a finite distance behind the eye, consequently the convex glass which corrects any definite amount of hypermetropia must be weaker the further it is placed from the eye. From this, too, it follows *that the effect of a convex glass of definite strength on hypermetropia is strengthened by removing it from, and weakened by approximating it to, the eye.*

The nature of the correction effected by a lens placed in front of an ametropic eye is therefore to give such a direction to parallel rays that they converge towards, or appear to diverge from, the far point of the eye. This is seen in Figs. 120 and 121, which represent respectively the correction of myopia by means of a concave lens, and of hypermetropia by means of a convex lens.

Numbering of lenses.—Lenses are numbered according to the length of their focus; that which has a focal length equal to 1 metre being now looked upon as having the unity of refractive power, positive or negative, and therefore being called 1·0, or −1·0. A lens of this power is called, too, a lens of 1 dioptre, or shortly, 1 D. One only half the refracting power of a 1 D. lens, and which has therefore a focal distance of 2 metres, is numbered 0·50, and one of twice the power, *i.e.*, with a focal distance of ½ metre, 2·0, or 2 D., and so on.

Lenses used as spectacles seldom require to be stronger than 20 dioptres, positive or negative—that is, 20 times as strong as a +1·0 or −1·0 lens, and having 1 D. more refracting power than a No. 19·0, 2 dioptres more refracting power than a No. 18·0, and so on.

Every unit of difference corresponds, then, to an equal increase or decrease in refracting power.

The usual trial cases contain the following spherical lenses, both plus and minus:—

0·25
0·50
0·75
1·0
1·25
1·50
1·75
2·0
2·25
2·50
2·75
3·0
3·50
4·0
4·50
5·0
5·50
6·0
7·0
8·0
9·0
10·0
11·0
12·0
13·0
14·0
15·0
16·0
17·0
18·0
19·0
20·0

In all, sixty-four different spherical lenses. In most cases there are two of each number.

For those who are not constantly working at the correction of errors of refraction, such a large number of trial lenses is unnecessary. By the combination of two lenses when required, any number from − 15·0 to + 17·0 can be obtained at intervals of 1 D. apart with the following nine lenses:—

− 5·0	+ 1·0	+ 4·0
− 10·0	+ 2·0	+ 8·0
− 15·0	+ 3·0	+ 13·0

Supposing, now, an eye to be focussed for parallel rays, either owing to its own formation or as the result of an optical correction such as we have considered, it will, when at rest, receive no distinct image of objects near at hand. All that is necessary, however, in order that the retinal image of any object at any distance should be distinct, is that the rays proceeding from it should be given such a direction as if they came from a distance, *i.e.*, that the greater or less divergence of such rays should be converted into parallelism. This could obviously be done by placing a convex lens in front of the eye, the focus of which lens coincided with the position of the object, and whose strength would therefore have to be less the nearer it lay to the eye. To take an instance:—An object lies at the distance of ¼ metre from the eye—the eye is, however, only focussed for distant objects; what lens must be placed in front of it, so that the near object may be seen distinctly? The lens whose focal distance equalled ¼ metre, or a lens of 4·0 dioptres, would evidently approximately do so, and the more accurately the nearer it lay to the eye. But as it could not actually be placed within the eye, its action would always be a very little too weak. Practically it would come to be placed from 15-20 millimetres in front of the position in which it would produce its greatest possible effect; so that instead of rendering the rays diverging from ¼ metre or 250 millimetres in front of the eye parallel, it would produce parallelism of rays emanating from a point 265-270 millimetres in front of the eye. The true correcting glass in the case of the ¼ metre distant object would evidently then be one whose focal distance was 235 millimetres, or less, instead of 250 millimetres. Now, what lens has a focal distance of 235 millimetres? We have seen that focal distances and the number given to lenses are reciprocal quantities; if we calculate in fractions of a metre, therefore, by dividing 235 into 1000, we should get the number of the required lens: this gives approximately 4·25. Obviously, the nearer the object which has to be seen distinctly lies to the eye, the greater would be the difference between the strength of the lens which would be required to be added to the refractive power of the eye itself, and that which might produce the same effect on being placed outside it. So that, taking the same distance of 15 millimetres as that at which the lens would lie in front of the point within the eye from which the measurement is made (a point whose position and importance will be afterwards considered), there would be a difference of one whole dioptre so soon as 15 millimetres made the linear difference between the focal distances of the two lenses. This is already the case with lenses rather less than 8 and 9 dioptres strength, while an addition of 14 dioptres to the crystalline lens itself would be equivalent to that of nearly 18 dioptres placed in the position in question in front of the eye.

It will now be apparent that should the eye not of itself be so formed as to focus parallel rays on the retina, the glass which renders this possible, and which therefore either diminishes or increases its refractive power, must be subtracted from or added to the lens otherwise required to give a distinct image of an object at any particular distance. Thus, if a myopic individual requires a $-2·0$ lens to see a distant object distinctly, he will require, as long as the refracting power of the eye remains unaltered, a lens of $4·0 - 2·0$, or $+ 2·0$, to focus distinctly an object at a little more than $\frac{1}{4}$ metre distant. On the other hand, a hypermetropic individual who can only see distant objects distinctly with a $+ 2·0$ lens, would require one of $4·0 + 2·0$, or $+ 6·0$, to focus an object of a little more than $\frac{1}{4}$ metre distance. *A weaker lens is therefore required for the focussing of near objects in myopia; and a stronger lens in hypermetropia than is required in emmetropia.*

ACCOMMODATION.

So far we have supposed the eye to be constantly exhibiting the same degree of refraction, that which results from the relative positions and curvatures of its various refractive media when uninfluenced by any circumstance by which these may be modified. The eye is, however, able by a muscular effort to increase the strength of its refracting power, or, in other words, to *accommodate* itself for the vision of near objects. This is effected by an increase in curvature of the surfaces, and consequent lengthening of the antero-posterior diameter, of the crystalline lens. It is mainly the anterior surface whose curvature alters. The effect of this is to add a convex meniscus to the lens, the refractive strength of which meniscus gives the measure of accommodation, so that we talk of 2, 3, 4, 10, or 14 dioptres of accommodation, according to the power of this additional lens within the eye; that is to say, that when, for instance, an eye alters its accommodation from that required for seeing an object at a distance to that required for focussing accurately one at $\frac{1}{10}$ metre, it has altered its power of refraction to exactly the extent which would correspond to the placing of a 10 dioptres glass lens (whose thickness was neglected) immediately in contact with the crystalline lens. The eye is then said to have exerted 10 dioptres of accommodation.

We have seen that the point for which the eye at rest is focussed is called the *far point.* The point, on the other hand, for which the eye is adapted when exerting its full power of accommodation—that is to say, the point which in the strongest state of refraction of the eye has its conjugate focus on the retina—is called the *near* point.

The focal strength, again, of that addition to the lens which brings the adaptation from the far to the near point, measures the *breadth* or *range of accommodation* in each case. Thus, if A denote the value in dioptres of the accommodative change, and F and N respectively the values of the lenses in dioptres whose focal distances measured from the eye would coincide with the far and near points of the eye, we have $A = N - F$.

It is evident that equal *ranges* of accommodation may coexist with different positions of far and near points. Thus there is the same change in refractive power produced by an addition of 5 dioptres within an eye focussed for parallel rays, as there is by the same addition of 5 dioptres in the case where it is already focussed for a distance of $\frac{1}{5}$ metre. Yet the linear distance between the two points focussed for differs very considerably, reaching, in the first case, from an infinite distance to 20 centimetres or 8 inches from the eye, in the second, from 20 centimetres to 10 centimetres, a linear difference of 4 inches. In both cases, A, the range of accommodation $=5{\cdot}0$ in the first, because $N=5{\cdot}0$ and $F=0$; and in the second, because $N=10$ and $F=5{\cdot}0$.

The accommodation is said to be *positive* when the eye, from being focussed for a more distant object, is adapted for the vision of a *nearer* one; *negative*, when the change in accommodation takes place in the opposite direction, and the refractive power of the eye is diminished. A positive change in accommodation is occasioned by a muscular effort; a negative change, on the other hand, is a result of a cessation of more or less of the muscular contraction, so that the eye in a state of rest, as far as muscular effort is concerned, is focussed for its far point.

Helmholtz, who first discovered that this alteration in the curvature of the surfaces of the crystalline lens occurred during accommodation, and was sufficient to account for the change of refraction produced, also gave the following explanation of how these alterations are brought about. In the position of rest, the lens is compressed in the direction of its axis, and more particularly on its anterior surface, by the fibres of its suspensory ligament (the zonule of Zinn), which are under a certain degree of tension. When this tension is released, the lens, owing to its elasticity, or, more correctly, the elasticity of its capsule, becomes thicker, the curvature of its anterior surface undergoing a very considerable increase, whilst there is a simultaneous slight increase in the curvature of its posterior surface as well. The cause of the release of the tension on the fibres of the zonule is the contraction of the ciliary muscle. When this muscle contracts it approximates the two ends to which it is attached, viz., the choroid on the one hand, and the tissues forming the angle of the anterior chamber on the other. In this way the position of attachment of the zonule fibres is drawn forward, and the tension which they exert on the lens capsule diminished. The retraction of the anterior attachment of the ciliary muscle causes a deepening of the peripheral portion of the anterior chamber during accommodation, whilst at the same time the central portion of the chamber is shallowed by the bulging forward of the anterior surface of the lens, and the consequent slight advancement of the pupil. During accommodation there is also a contraction of the pupil.

It follows from what has been said with reference to the mechanism of accommodation that this power is entirely absent when the lens is absent from the eye.[1]

The range of accommodation, depending, as it does, on the extent to which the curvature changes take place in the crystalline lens, in response to the action of the ciliary muscle, is influenced by the consistency of the lens substance, as well as by the functional activity of the ciliary muscle or the nerves which supply that muscle. Diminution in the range of accommodation, due to changes which take place in the lens, is physiological in so far as such diminution slowly and gradually takes place during life; probably not beginning, however, till after the

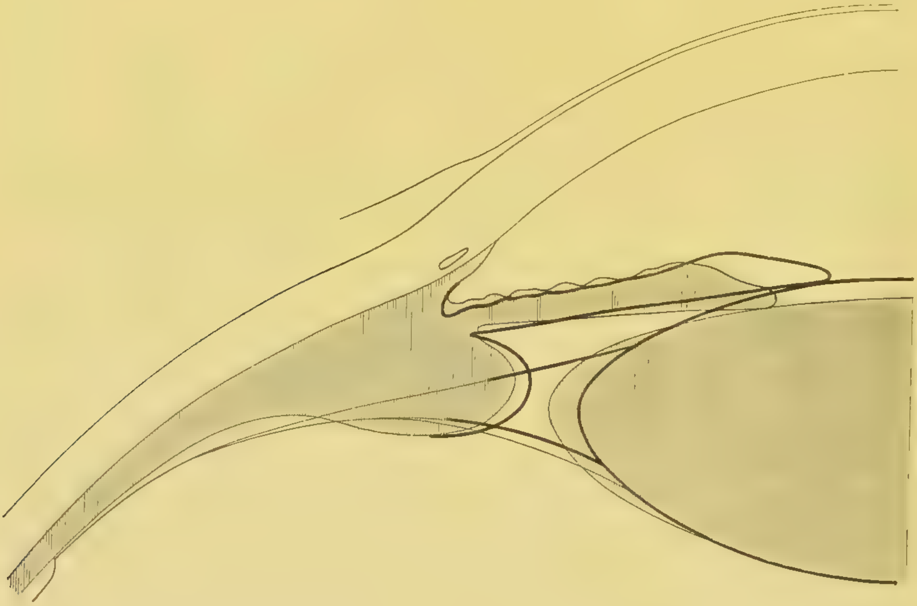

Fig. 122.—Diagram showing position of lens, &c., in eye at rest and in accommodated eye (after Fuchs).

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[1] For further particulars in connection with accommodation, as well as a discussion of the experiments on which this explanation is given, the reader may be referred to Helmholtz's *Phys. Optics*, second edition, page 12.

first few years of childhood. Although, therefore, not absolutely identical, there exists a very similar range of accommodation in all individuals of the same age, the range being less the older the individual, until it completely disappears, and no power remains of altering the refraction of the eye.

The linear distance separating the far and near points—or, shortly, *the position of the range of accommodation*—depends, on the other hand, upon the state of refraction of the eye; but it, too, may change with age, owing to the tendency which the condition of refraction has to alter at different times, more especially up to the period of full growth, and after the age of sixty.

The most favourable position of the range of accommodation is that which admits of the focussing of objects through the greatest distance. This is what occurs in emmetropia, and in cases of ametropia where the ametropia is fully corrected. In hypermetropia, when the range of accommodation is considerable, and the hypermetropia not of high degree, the position of the range of accommodation is also favourable, *i.e.*, although a certain portion is required for the adaptation of the eye for distant vision, there still remains a sufficient amount to enable it to focus objects at a distance near enough for all practical purposes. The position of the range of accommodation is most unfavourable in myopia, as in no state of active or passive refraction is it possible for the unaided eye to get properly distinct images of distant objects, so that its accommodative range only serves the purpose of adapting it for vision of near objects,—the linear distance corresponding to the same accommodative range being of course smaller, and consequently less serviceable, the higher the degree of the myopia. Indeed, even when the myopia is by no means considerable, say 4 dioptres in amount, the range of accommodation which the eye possesses is, unless the error be corrected, of little practical use, as few objects require to be looked at, at a shorter distance than $\frac{1}{4}$ metre. Myopia of that degree, at a time of life when there exists a range of accommodation of say 10 dioptres, is associated with greater inconvenience, so far as the possibility of receiving distinct images of objects at different distances is concerned, than a hypermetropia of the same degree, and with the same accommodative range, 6 dioptres of which, after 4 dioptres have been employed to adapt the eye for parallel rays, cover the distance from the most distant objects to those only $\frac{1}{6}$ metre from the eye.

It will now be readily understood that more is usually effected by the correction of ametropia than the mere optical adaptation of the unaccommodated eye for the focussing of parallel rays. In all cases where a fair range of accommodation still remains, the position of the range is thereby at the same time displaced, so as to become as favourable as possible.

The contraction of the pupil, which occurs along with efforts at accommodation and convergence of the visual axis, is of more or less service in extending the range of distinct vision. On account of the smallness of the pupil and consequent diminution in the size of the circles of diffusion, the position at which ordinary sized print, such as that used in newspapers, can be seen, lies considerably within the real near point, or that for which the eye is actually focussed.

The determination of the range of accommodation (A) in a large number of individuals of different ages was made by Donders, who obtained an average for different ages from which he plotted a curve (Fig. 123) in whichthe ordinates represent the age and the abscissæ, the corresponding ranges of accommodation measured in dioptres. This curve enables us to see at a glance what is the average range of accommodation for any age and conversely gives for a certain range of accommodation the average age at which that range is found.

The curve gives, too, the actual position of the near point corresponding to any age, supposing the eye to be emmetropic. By merely displacing the curve upwards or downwards, the position of the near point at the same age (and indicated in the same way by the number of the dioptre lens having an equivalent focal length) for any degree of myopia or hypermetropia is obviously also given, as it is clear that this must be got by merely adding or subtracting the value of the far point lens. Thus in myopia of 5·0 dioptres according to the diagram at ten and twenty years of age, the distance of the near point from the eye would be respectively represented by the focal distance of a lens of $14{\cdot}0 + 5{\cdot}0 = 19{\cdot}0$ and $10{\cdot}0 + 5{\cdot}0 = 15{\cdot}0$ dioptres,—*i.e.*, it would be in the first case $\frac{1}{19}$ instead of $\frac{1}{14}$ of a metre distant from the eye (a linear difference of about 19 millimetres), and in the second $\frac{1}{15}$ instead of $\frac{1}{10}$ metre from the eye (a linear difference of over 33 millimetres). And whilst in ametropia the near point has usually at about the age of sixty-four receded so as to be infinitely distant, an individual whose myopia at that age was 5·0 would still have a near point rather within $\frac{1}{5}$ of a metre from the eye. Again, taking a case of hypermetropia 5·0; we should have the average near point at ten and twenty years represented respectively by lenses $14·0 - 5·0 = 9·0$ and $10·0 - 5·0 = 5$. Before forty the near point would have receded to infinity, as by that time in emmetropia it is represented by rather less than 5·0, and in the degree of hypermetropia we are considering would be rather less than $5·0 - 5·0 = 0$.

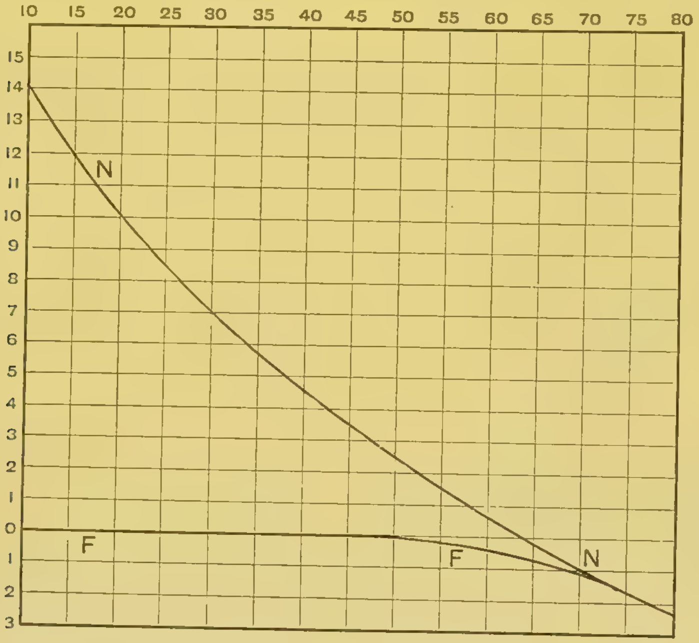

FIG. 123.—Donders' curve of range of accommodation (after Nagel).

Practically, it has to be remembered that whilst the curve of Fig. 123 gives the average, we may frequently find not inconsiderable real or apparent differences, more especially in the direction of the range being greater than is there given. It has to be remembered that the near point, and this refers more especially to young people, may not always appear the same in the same case, as much depends on the effort exerted to increase the refractive power, and the greatest possible effort can only be momentarily maintained.

One of the simplest practical methods of finding the position of the near point in any case is to determine the strongest concave glass with which the individual can see distinctly small print (of a size corresponding at a definite distance from the eye to something approaching the limit of his visual acuity); or, where the near point lies beyond the distance at which the print is held, the weakest convex lens with which it can be seen. In order to do so he has, of course, to add as it were an equivalent convex lens to his eye in order to overcome the concave one placed in front of it, so that the strength of that addition, and consequently the point for which the eye is focussed when exerting its full amount of accommodation, *i.e.*, its near point, can be readily calculated from the strength of the concave lens and its distance from the eye. This is seen in Fig. 124, where the meniscus-shaped portion of the crystalline lens is that which counteracts the effect of the concave lens.

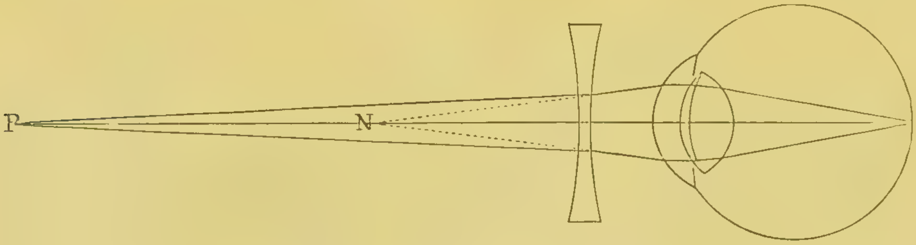

Fig. 124.—Showing the manner in which the action of a concave lens can be overcome by alteration in the curvature of the crystalline lens.

In order to exert the greatest possible amount of accommodative power, most people have to bring about a degree of convergence of their visual axes, which is greatly in excess of that which would direct the lines of vision on the nearest point thus accommodated for. What is called the *absolute* near point can therefore, as a rule, only be attained by the sacrifice of binocular vision. The binocular near point and the binocular range of accommodation have been distinguished by Donders from the absolute. It is seldom that the difference between the two amounts to anything of practical importance.

Donders, to whom we owe in great measure our knowledge of the nature of accommodative and refractive errors, has also pointed out the importance of what he has called the *relative* range of accommodation. This may be looked upon as the range of accommodation which is at the disposal of the individual for any particular degree of convergence; that is, given any point on which the visual axes converge, the difference between the furthest and the nearest point for which the eyes can be accommodated without changing the point of fixation.

The relative range of accommodation varies for different distances of fixation; but the practical importance to be attached to it is usually limited to the amount and disposition of the range which exists for the reading distance. That portion of the relative range which consists of the difference between accommodation for the distance fixed, and the nearest point for which accommodation with that degree of convergence is possible, is called the *positive* portion of the relative range of accommodation. That portion, on the other hand, which is the difference between the degree of accommodation for the point fixed and the furthest point which, under the circumstances, can be distinctly focussed for, is called the *negative* portion. The positive portion is determined by the strongest concave glasses which can be overcome, and the negative by the strongest convex glasses which can be overcome without any change in convergence.

It is generally considered that only when the positive portion is at least as great as the negative is the distance one for which accommodation can be maintained for any length of time, without giving rise to pain or discomfort, or, in other words, to what is called *accommodative asthenopia.* In some cases, however, there is a considerably greater than normal negative relative accommodative range, so that probably the proportion between the negative and positive portions is of less importance as affecting the capability of maintaining the necessary degree of accommodation for the working distance, than the actual amount of the positive portion, which should at least equal 2·50 to 3·0 dioptres.

The direction of fixation appears also to be of some influence on the range of relative accommodation. It is not always the same, for instance, in looking down as on looking up at equally distant objects. The difference in this respect is subject, too, to individual variation.

Fig. 125 is Donders' curve of relative accommodation in the case of an emmetrope aged fifteen. The equally distant horizontal lines represent intervals of 1·0 dioptre of accommodation, so that the point where they cut the curve gives the amount in dioptres of accommodation exerted. The vertical lines represent the angles of convergence made by the two visual axes corresponding to the distances for which the eyes are accommodated. The numbering is according to Nagel's metre angle notation, which is based on exactly the same system as that explained for the metrical numbering of lenses. According to this notation, the angle which the visual axis of each eye makes, when a point in the middle line is fixed, with the direction they have when fixing a distant point straight in front of them, is expressed as the reciprocal value of the distance from the eye to the nearer point measured in metres, or fractions of a metre. Thus, if the distance fixed be $\frac{1}{3}$ metre, the convergence is said to be 3 metre angles in amount; if $\frac{1}{10}$ of a metre, distance 10 metre angles, and so on. Similarly the difference in the degrees of convergence between the direction required for fixation of a

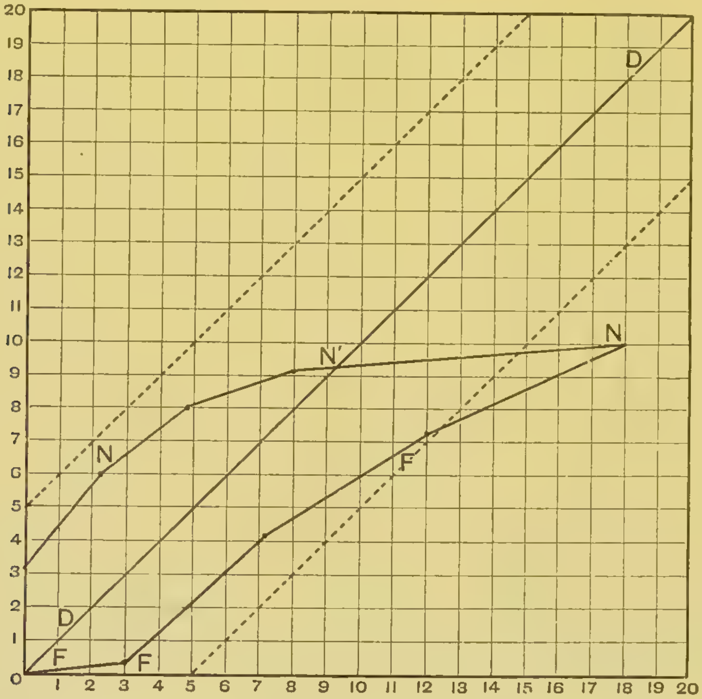

Fig. 125.

point $\frac{1}{3}$ of a metre, and another $\frac{1}{10}$ of a metre from each eye, equals 7 metre angles.

The normal or ideal relation between convergence and accommodation, in which, for any number of dioptres of accommodation exerted a similar number of metre angles of convergence are described, is represented by the diagonal line. This relation can obviously only exist when the refraction is emmetropic, and the position of rest of the visual axes parallelism, or where a definite degree of hypermetropia or myopia is associated with a corresponding amount of divergence or convergence of the axes in the state of rest. The position of any point on the curve above the diagonal represents the positive, that of any point below the diagonal, the negative portion of the relative accommodation for the degree of convergence represented by the vertical line which passes through it. In the case for which the curve is plotted we see that binocular vision has been impossible for a point N′ lying nearer than between $\frac{1}{9}$ and $\frac{1}{10}$ metre from the eyes, and that although 18 metre angles of convergence were possible, this did not result in an absolute near point closer than $\frac{1}{10}$ metre. The curve also shows that even although it has been impossible to accommodate for the nearest points for which the eyes were converged, still a certain range of relative accommodation remained up to the greatest possible convergence. Here, too, we see that this positive portion of the relative accommodation range has equalled the negative up to a convergence of 5 metre angles, so that the individual has been in the position of being able to maintain the amount of accommodation necessary for reading at $\frac{1}{5}$ of a metre, or, say, 8 inches, without any inconvenience.

If the myopic or hypermetropic individual were situated exactly as the emmetropic in respect to the association of the convergent and accommodative movements,—that is, if his zero point of convergence corresponded to his zero point of accommodation—it is evident that, when the ametropia was at all considerable, he would have to sacrifice binocular vision in order to see distinctly. We have only to displace the diagonal line downwards in the case of myopia, and upwards in the case of hypermetropia, to see at a glance what the requirements of these states of refraction are, and how they become more difficult of attainment on the supposition made, the higher the degree of ametropia. Thus the lower line represents the requirements of a case of myopia of 5·0 dioptres; it shows that up to a 5 metre angle of convergence no accommodation is required, whilst for 8 metre angles of convergence, for instance, only 3·0 dioptres of accommodation are necessary. Again, a glance at the upper dotted line of the diagram, which represents the requirements of a case of hypermetropia of 5·0 dioptres, will show that up to 5·0 dioptres of accommodative effort the visual axes must remain parallel, whilst for 8·0 dioptres of accommodation only 3 metre angles of convergence are required.

The actual state of matters in cases of ametropia is, however, not so bad as it would be, did the conditions as they exist in emmetropia hold good in the manner we have supposed. There is a great difference in this respect in different cases, a difference which depends partly on the extent to which the nature of the association of the two impulses to convergence and accommodation differs from that which we have looked upon as normal or ideal, and partly on the range of relative accommodation.

If the associated impulses were always connected as in emmetropia, we should expect to find that as soon as one eye was occluded from vision of the object fixed, it would take up a position which brought the axes of vision to bear on the point accommodated for. In the case of myopia, then, the occluded eye should diverge, and in the case of hypermetropia converge. This is, however, by no means invariably the case; when it is, the degree of deviation is indeed, as a rule, not co-extensive with the requirements of an intimate association.

The starting-point of the convergent effort, it must be borne in mind, is not always parallelism; that is to say, the position of rest or equilibrium of the two eyes may be a divergent one, and this may influence the position taken up by the occluded eye, under the circumstances just referred to. But even when this position of equilibrium is allowed for there still remains such a degree of disagreement between the results afforded by this simple experiment and those which the ideal association would necessitate, that there can be no doubt the association is often of another nature, altogether more favourable, as a rule, to the individual case.

Associated movements, that is to say, movements associated in such a way that a voluntary stimulus to the one liberates an involuntary stimulus to the other, may be either congenital or acquired. Of what nature the two under consideration are, has been, and still is, a disputed point, some holding that the exigencies of every individual case lead to a greater or less dissociation of accommodation and convergence, which are naturally intimately connected in the ideal manner, while others again believe that the two originally independent movements become associated in such a manner as to meet the requirements of each case. Which of these two views is the correct one, is a matter perhaps of small importance. The practical point is that the nature of the association is very variable, both in respect to the relative points of departure of accommodative and convergent movements, and the closeness or laxity of the existing relation between them.

Two circumstances influence, no doubt, the conditions of association, viz., heredity and tendency which the state of refraction has to change. Thus, with regard to the first, there are many points which seem to indicate that an individual whose ametropia is undoubtedly inherited is less likely to be unfavourably situated in the association of his accommodative and convergent efforts, than one in whom the error of refraction is more distinctly accidental, whilst, in the second place, much evidently depends on the rapidity with which the refractive changes take place, as when rapid a corresponding adaptation becomes less easy.

A full correction of the ametropia in any case, by which, as we have seen, the position of the range of accommodation is altered to what obtains in emmetropia, frequently gives rise to discomfort, owing to its introducing at the same time a less favourable situation of the existing relation between accommodation and convergence. This is more especially the case when the correction is made in adult life.

On the other hand, a full or partial correction often relieves the asthenopia, which an unsuitable relation in the associated impulses occasions.

A question which naturally suggests itself in connection with accommodation is, as to whether or not the two eyes can be accommodated to different extents at the same time. For objects seen in the middle line, and where the refraction is the same in the two eyes, an unequal degree of accommodation would not be required in order that the utmost distinctness of the images on both retinæ should result. On the other hand, when the object fixed is to the one side, and not far from the eyes, or where there is unequal refraction in the two eyes (what is called *anisometropia*) an equal amount of accommodation would obviously not permit of absolutely distinct images being obtained in both eyes. What then happens under these circumstances? Do the two eyes accommodate unequally, so as, when the difference is not too great, an equally distinct image is obtained in each eye? Or do they accommodate to the same extent, and if so, is one properly focussed, or does the accommodation take place in both for an intermediate distance? By holding a weak prism in front of one eye, with the base directed upwards or downwards, whilst a single line of small print or other suitable test-object is fixed, either well to the one side, or in the case of anisometropia in front of the face, it will invariably be found that the image corresponding to the one eye is perfectly distinct, whereas that which belongs to the other is more or less blurred, according to circumstances.

In anisometropia it is usually the eye for which the accommodative effort required for the distance fixed is smallest that is properly focussed. Therefore, when one eye is myopic and the other emmetropic, the myopic one is correctly focussed; when one is emmetropic and the other hypermetropic, the emmetropic one, and so on. In the fixation of latterly placed objects, it seems generally to be the eye which lies nearest to the object which determines the amount of accommodation to be exerted. In both cases much depends on the state of visual acuity of either eye, and on which of the two retinal images the individual is in the habit of unconsciously directing most attention, but the relation existing between the accommodative and convergent impulses is no doubt also of influence in this respect.

The contraction of the pupil which takes place along with accommodation is a muscular movement associated, in all probability, with both the accommodative and convergent movements. It is easy to show that even when accommodation takes place, while the direction of the visual axis remains unaltered, there is a corresponding change in the size of the pupil; but there exists all the time an impulse to convergence (which is at once rendered manifest by the position taken up when one eye is occluded), which is only restrained by the stronger impulse for the fusion of the binocular images, with which impulse the actual pupillary change may be connected.

PRESBYOPIA.

When the near point has receded beyond 22 centimetres, or a little more (about 9″), which the curve in Fig. 123 shows takes place at or about the age of forty-five in emmetropes, a difficulty is experienced in reading small print, more especially when badly illuminated, or in working at anything which requires to be taken near to the eyes in order to give a large enough image to be seen distinctly. To this defect in vision the name of *presbyopia* or "old sight" is given.

When an individual becomes presbyopic he is obliged to wear convex glasses in order to see distinctly anything which he has to take within about nine inches of his eyes. The glass which has to be worn under these circumstances should be of such a strength as to bring the near point to between 23 and 24 centimetres from the eyes, *i.e.*, it should supplement the defect in accommodation to such an extent as to make the combined strength of the accommodative change (measured from the existing adaptation for a distance) and the glass equal to that of a lens of 4·50 placed in front of the eye. In emmetropia, then, it must supplement the range of accommodation by just the amount that it falls short of 4·50 dioptres.

In hypermetropia, although there is for the same age the same average range of accommodation, the position of the range is unfavourable for near vision, and the hypermetrope is unable to bring small objects sufficiently near to his eye, *i.e.*, he becomes presbyopic at a time when he possesses a greater range of accommodation than 4·50 dioptres, and therefore at an earlier age than is the case in emmetropia.

For myopic individuals the reverse holds good—the position of their range of accommodation is favourable to near vision; the near point is consequently longer in receding to the extent which renders the aid of a convex glass necessary, so that presbyopia is later in making its appearance in myopia than in emmetropia.

From Fig. 123 it will be seen that the originally emmetropic eye becomes, as a rule, more or less hypermetropic after the age of sixty-five; this is represented by the dip of the far point curve, so that after that age stronger glasses than 4·50 may be required. The cause of this senile hypermetropia is similar to that which gives rise to the advancing diminution in the range of accommodation, viz., the sclerosis of the lens. This produces not only a loss of elasticity, but also a change in the refractive indices of the different layers of the lens. The layers become more and more dense, and eventually equal in their refractive power the central or nuclear portion. The result of this is to diminish the refractive power of the lens as a whole, and therefore of the eye.

The glass required for the correction of the presbyopia in a hypermetropic or myopic individual is at once determined from that required for an emmetrope of the same age by merely adding to or subtracting from it that which corrects the ametropia. Thus, if an emmetrope required + 3·0, a hypermetrope of the same age to the extent of 2·0 dioptres would, as a rule, require + 5·0, and a myope to the same amount + 1·0.

Measurement of Ametropia.

The determination of the degree of myopia or hypermetropia may be made both subjectively and objectively. With a little intelligence and good acuteness of vision on the part of the individual tested, the subjective method is the most rapid and accurate. It is only, therefore, when either of these factors is deficient, or when the patient tries for any purpose to deceive, that an objective test is called for.

The *subjective test* is made by determining the glass required to give the best vision at a distance. The distance should be at least five, preferably six metres, and the test objects be types or figures graduated in size, according to the method first adopted by Snellen.

If the patient's accommodation be paralysed by atropine, or be absent owing to advanced age, or any other cause, the concave or convex glass which enables him to read the smallest type which he is capable of, is a measure of the degree of his ametropia (referred to the position of the glass).

The presence of accommodative power renders it possible for him to see equally well (though not so comfortably) with different strengths of glasses. *This circumstance must always be kept in mind whilst making a subjective test under the ordinary conditions.*

In the case of myopia, the degree of normal refraction is given by the *weakest* glass with which the individual obtains his best vision (provided there is not any marked defect in his acuity). Stronger glasses do not necessarily diminish the acuteness of his vision, because they may be, and in the case of young myopes almost invariably are, neutralised or overcome by an accommodative change in the crystalline lens.

In the case of hypermetropia accommodation plays a still more important part. We have seen that this condition of refraction, when not exceeding in amount the amplitude of accommodation at the disposal of the individual, may be corrected for by accommodation. This is, as a rule, what is done, more or less of the amplitude of accommodation being brought into play in order to permit of distinct vision. The desire to see anything distinctly, which is almost constantly present when surrounding objects are sufficiently illuminated, is habitually associated, then, in hypermetropes with an accommodative effort. If, therefore, we put a convex glass in front of the eye, we can only expect to find that with it the hypermetrope, by relaxing a corresponding amount of accommodation, will maintain the same visual acuity which he had without it. *If, then, a convex lens either produces an improvement in vision or does not render distant objects less distinct, the eye must be hypermetropic.*

But we cannot, as a rule, determine the degree of hypermetropia by means of the convex glass held in front of the eye, in the same way as has been explained can be done with a concave glass in the case of myopia. The reason of this is that the accommodation brought into play in order to overcome the refractive error cannot be altogether relaxed during attempts at seeing, and thus a portion of the hypermetropia remains latent. The *total* degree of hypermetropia (Ht) is divided then into a portion which is *latent* (Hl), and a portion which is *manifest* (Hm); or $Ht = Hl + Hm$. The proportion between the latent and manifest portions of the hypermetropia varies with the age of the individual, and also to some extent with the degree of the total hypermetropia. The older the individual the smaller is the proportion which remains latent, so that after a certain age—from forty to forty-five—the whole amount is manifest. The lower degrees, too, are more persistently corrected by accommodative efforts than the higher.

The *strongest* glass, therefore, with which the individual retains the full degree of visual acuity which he possesses is the measure of his *manifest hypermetropia.* A determination of the whole hypermetropia subjectively is only possible when the accommodation is paralysed. A close approximation to it may be got by an objective examination.

When the fundus of an eye is illuminated by means of the ophthalmoscope, the rays which are reflected from any point in it proceed after refraction through the dioptric media, as if they diverged from or converged towards some other point, the image of the first. If the eye be hypermetropic, and the defect not corrected by accommodation, they will diverge as if from a point behind the eye; if myopic, they will converge to a point in front of it. The distance of this, the *far point*, measures in either case the degree of the ametropia.

The distance of the far point is generally measured from one of the cardinal points of the eye (*vid. infra*). For the theoretical consideration of refractive errors, it seems best to adopt the first principal point as that from which the far point is calculated. For most practical purposes, however, the anterior focus is the point from which it may be most conveniently measured, so that when the far point lies 10 centimetres behind the anterior focus the eye is hypermetropic to the extent of 10·0 D.; when 10 centimetres in front of the anterior focus, there is a myopia of 10·0 D. The advantages of this way of measuring the degree of ametropia are that it then corresponds to the strength of the correcting glass, and further (as is afterwards explained), in the case of axial ametropia, to the axial change.

In the emmetropic eye the rays reflected from a point in the retina pass after refraction through the dioptric media, not in the shape of a cone but as a cylinder, all the rays being parallel to the one which passes from the point on the retina to the nodal point. No image of any extent of the fundus of the emmetropic eye is formed in the observer's eye, unless the ophthalmoscope mirror be held very close up to the eye examined. If held any distance off nothing is seen but a red reflection. The reason of this is that the cylindrical bundles of rays from the points at either extremity of a portion of the retina cross between the eyes of the observer and observed, and thus do not pass through the aperture of the mirror, so that what is seen is merely a collection of rays from a very small area, not sufficiently extensive to be recognised in structure. The larger the pupil of the eye examined the greater is the area from which rays pass into the observer's eye, consequently the larger is the portion of the fundus which he can see at the same time; and, given a definite size of object—such, for instance, as the optic disc—the farther away it can be seen.

A distinct image of a portion of the fundus of a myopic eye is formed in the observer's eye, if the ophthalmoscope mirror be held at a distance greater than that of the far point of the myopic eye by the distance of the observer's near point. The amount of the fundus whose image is thus seen depends on the size of the pupil and on the degree of the myopia. The larger the pupil the greater the image with a given degree of myopia. Again, the higher the myopia the greater is the portion of the fundus seen with a given size of pupil. When the observer's eye is nearer the eye examined than its far point, it receives rays from a larger portion of the fundus, but, unless his eye be hypermetropic, these do not give the observer a clear image. The reason of this will be explained further on.

A distinct image of a portion of the fundus of a hypermetropic eye is received by an observer reflecting light into the eye from some distance. The amount of the fundus seen depends on his distance from the eye, on the size of the pupil, and the degree of the hypermetropia. It increases as the distance separating the two eyes diminishes, and as the pupil and degree of hypermetropia increase.

If, therefore, the observer at some distance from the eye receives with the ophthalmoscope mirror alone a tolerably clear view of any part of the fundus, such as the vessels or optic disc, the eye examined must be ametropic.

If the patient move the eye slowly in any direction, the image seen will appear to move, and the direction of the movement at once shows whether the fault in refraction is one of myopia or hypermetropia. In myopia, the image will move in the same direction as the eyes; in hypermetropia, in the opposite direction.

This is evident, because in myopia it lies between the observer's and the patient's eye; therefore, in the first case, being in front of the centre of rotation, it moves with the anterior part of the eye.

If, instead of the patient moving his eye, the observer moves his own head, still keeping the fundus in view, then in myopia the image moves in an opposite direction to that of the observer's head, while in hypermetropia it moves in the same direction.

The movement here observed is a parallactic one, due to the position of the images of the fundus with respect to the pupil. As the pupil lies behind the image in myopia, and in front of it in hypermetropia, their apparent movement is in opposite directions, the more distant object always appearing to move with the mirror.

Another objective method of arriving at the diagnosis of the condition of refraction is to observe the change which takes place in the size of the optic disc, as the lens held between the ophthalmoscope and the eye examined, in the indirect method of ophthalmoscopic examination, is approached or withdrawn from the eye. If the eye be myopic, the withdrawal of the lens causes the image of the disc to increase in size; if hypermetropic, to diminish, while in the emmetropic eye no alteration in the size of the disc takes place on changing the position of the lens.

The principal value of this method is in the objective diagnosis of astigmatism, as will be afterwards explained. In ametropia, if it be high, it is also observable; but in low, or even moderate degrees, the change which takes place is apt to escape detection, as the *apparent* size depends on the distance to which the image is projected in space. The change in size for different positions of the auxiliary lens is shown in Fig. 126 in the case of myopia and hypermetropia. In the figure, *obj* represents the image of the disc, or other part of the fundus, which is formed by the eye, and which is therefore in its turn the object whose image is formed by the lens. As some of the rays used to find the position of the image do not actually pass from the eye, they are represented as dotted lines.

The indications afforded by the mirror alone, or by the indirect method of ophthalmoscopic examination, are only just sufficient to enable one to diagnose whether the refraction is greater or less than normal.

An approximation may be made to the degree of the ametropia in various ways. Two methods are in general use for thus estimating the refraction, viz., the examination of the fundus oculi with the ophthalmoscope by the direct method and the shadow test. The first is the more trustworthy method, unless the patient's accommodation has been paralysed with atropine. It requires, however, some practice, as, in order to give the nearest approximation, it is necessary for the observer either to relax his own accommodation thoroughly, or to be

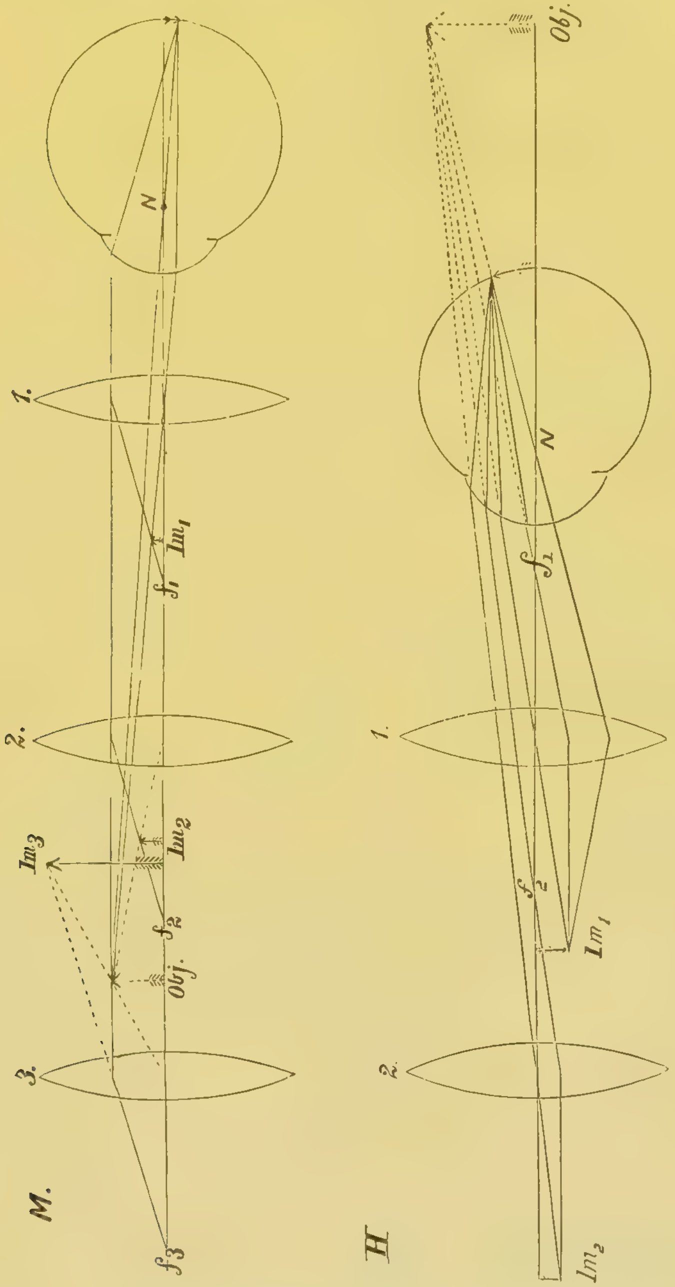

FIG. 126.—Showing the alteration in the size of the ophthalmoscopic image as the lens is withdrawn from the eye in myopia and hypermetropia.

aware of the extent to which he is exerting it. It is of course essential that the patient's accommodation should also not be brought into play, but there is as a rule not much tendency to this, as the examination is made in the dark, and there should be no near object to attract his attention.

In order to be able to estimate the refraction of another individual, the observer must, in the first place, be conscious of the state of his own refraction. The glass which corrects this, when it is not emmetropic, is brought in front of the aperture of the ophthalmoscope, and forms the starting-point from which any other glass which may be required to obtain a distinct image of the patient's fundus is calculated, as representing the one which in the position which it occupies in front of the patient's eye approximately corrects his ametropia. Thus, if the details of the fundus appear perfectly distinct to the observer when examining with the ophthalmoscope close up to the patient's

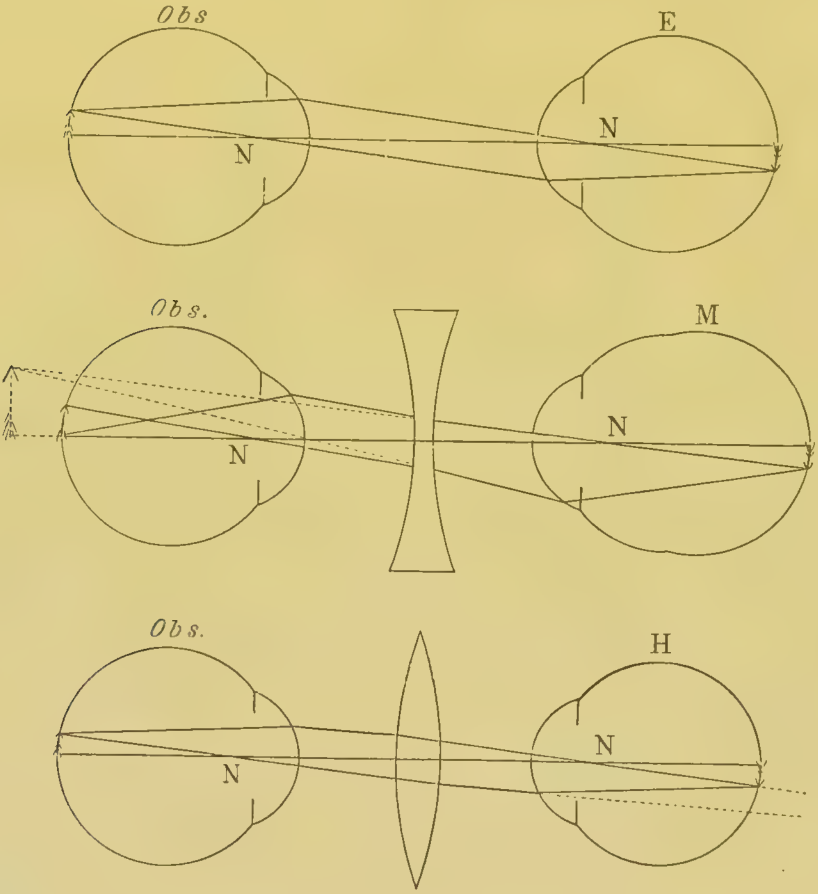

FIG. 127.

eye, the accommodation of both eyes being relaxed, and either without any glass intervening between his own eye and the aperture in the mirror, or with that glass which corrects an existing error in his own refraction, then the patient must be approximately emmetropic, as the rays proceeding from any part of the fundus are then all parallel to that which is directed to the nodal point, and such a parallel bundle of rays must, after refraction through the observer's eye, or through the system of correcting lens and eye, meet at some point on his retina. In Fig. 127 E, the course of the rays is shewn in the case of emmetropia of both observer and patient.

When the patient is not emmetropic it is necessary for the observer, always supposing that he keeps his accommodation relaxed, to bring such a lens behind the ophthalmoscope as shall focus his own eye for the far point of his patient. Thus, if the patient be myopic, he must render his own eye hypermetropic to an equivalent extent, as the rays which meet his eye from any point on the fundus of the patient are convergent instead of parallel, and therefore meet at a point in front of his retina. The concave lens necessary to effect this is that which will correct the patient's myopia, the determination of the degree of which can therefore be made in this way. This is represented at M, Fig. 127.

If the patient be hypermetropic, the observer must bring in front of the ophthalmoscope aperture a convex lens, whose focal distance corresponds to that of the patient's far point, in order that the divergent rays from all points on the patient's retina may be rendered parallel, and thus be brought again to a point on the retina of the observer. The strength of the lens which effects this is, of course, the measure of the degree of the hypermetropia when corrected in the position occupied by it in front of the eye. This is represented in Fig. 127, H.

The estimation of refraction by means of the shadow test is made by observing the direction of motion of the red reflection, got by throwing light into the eye from an ophthalmoscope mirror held at some distance and rotated slightly in a definite direction, while lenses of known strength are placed in front of the eye. It has been called the shadow test, because attention is directed perhaps more to the dark shadow which borders the illuminated area than to the area itself.

The examination is made in the following way. The room must be darkened. From the source of light, which may be placed either at the side of the patient and shaded from him, or immediately over his head, a beam is reflected by means of an ophthalmoscope mirror. The mirror may be either plane or concave, but, other circumstances being similar, the movements made by the shadow are in opposite directions, according as one or other is used. Thus, if in any case the shadow moves in the same direction as that in which a plane mirror is rotated, it will move in the opposite direction to the rotation of a concave one. The reason of this will be afterwards explained. In the meantime, as concave mirrors are most generally used, the appearances met with when such a mirror is employed will be described. The observer should be seated 1 metre from the patient. If on slightly rotating the mirror not too quickly in a horizontal plane, the shadow appears to move in the same direction as the rotation, or "with the mirror," then the refraction is myopic. If it move in the direction opposite to that in which the mirror is rotated, or "against the mirror," the refraction is either emmetropic, hypermetropic, or only slightly myopic. By putting lenses in front of the eye—concave if the direction of the shadow indicates myopia, convex if it indicates hypermetropia—we may arrive at a strength of the lens in either case which reverses the direction of the shadow, and therefore get a first approximation to the lens which is required for the correction of the ametropia if it exists. The weakest lens which reverses the movement, from being with the mirror to being against the mirror, is not quite the measure of the myopia. On the other hand, the weakest lens which reverses the movement from being against the mirror to being with it, is rather higher than that required to correct the existing hypermetropia. If the lens is not higher than + 1·0, the refraction cannot be far from emmetropic.

The shadow test has received many names, *e.g.*, keratoscopy, retinoscopy, koroscopy, skiascopy.

The explanation of the appearances met with in the different states of refraction is as follows:—When light is thrown into the eye from any source, the size of the illuminated area of the fundus will depend on the distinctness of the image of the source of light on the retina, while the position of the illuminated area will alter with that of the source of light, and move in a direction opposite to that in which the illuminating source is moved. When a plane mirror is used to reflect light into the eye, the direction of the rays, after reflection, is such as if they came from some source behind the mirror. This apparent source is displaced in the opposite direction to that in which the mirror is rotated, and consequently the area of the fundus which it illuminates is displaced in the same direction as that in which the mirror is rotated. When a concave mirror is used, the apparent source of light lies somewhere in front of the mirror, or between it and the eye, where an image of the source is formed, and this apparent source is displaced in the same direction as that in which the mirror is rotated. On this account the illuminated area of the fundus is displaced in the opposite direction. What is seen, however, by the observer is not the illuminated area itself, but its image, as formed by the eye. This image is erect if the rays from every point of the area meet behind the eye, or so far in front of it as to be behind the eye of the observer; the image is inverted if the rays meet before they reach the observer's eye. In the first case, where the refraction is hypermetropic, emmetropic, or only slightly myopic, the movement of the illumination, or the shadow which borders it, is in the same direction as that which actually takes place in the eye, *i.e.*, as we have seen, against the mirror. In the second case, it is in an opposite direction to that taken by the area of illumination at the back of the eye—that is, it is "with the mirror."

The neutral point corresponds, of course, to a coincidence of the far point of the patient's eye with the plane of the observer's pupil. When, therefore, the observer is 1 metre from the patient, and the patient's eye is myopic to the extent of 1·0 D (and not accommodated), the direction in which the illuminated area moves as the mirror is rotated cannot be seen. There is then such a gradual transition from light to darkness in the area of illumination that the dark shadow corresponding to where the illumination stops altogether is not appreciated. Practically, therefore, what has to be determined in every case is the glass which causes the reversal of the shadow, and not that which exactly corrects for the distance of a metre. On this account, too, the examination has to be made at a distance, and for purposes of calculation 1 metre is a convenient distance. Small differences in the actual position of the patient's far point are not appreciable. Whether it is 5 centimetres in front of, or 5 centimetres behind the observer's pupil makes but little difference in the estimation of the refraction at 1 metre. The possible error from this source is at most $\frac{1}{10}$ dioptre. But the error is increased in an increasing ratio for nearer and nearer distances between the eyes of observer and patient. It is already considerable when the eyes are separated by as much as a foot.

The brightness of illumination, and, as a consequence, the distinctness of the shadow, is less when the ametropia of the patient is great than when his far point either lies near, or is brought by means of an intervening glass to be near, the observer's eye. This increase in the distinctness of the shadow, as the proper correction is approached, is a useful guide in the practical determination of the refraction by the shadow test. The reason of the difference in brightness is that in ametropia the circles of diffusion of the image of the source of light are large compared with the visual angle under which the light is seen.

Cardinal Points.

Many problems in connection with refraction are much facilitated by the knowledge of the position of certain imaginary points, which have received the name of cardinal points. The properties and positions of these points may now be discussed.

Having regard to the degree of approximation which we have assumed, *i.e.*, writing for the tangent of the angle of divergence of the rays meeting any surface the angle itself, we may look upon the tangent plane to the surface as coincident with the surface. So that as US and SV (Fig. 128) come to equal UH and HV in the limit, we may consider the direction of the ray after refraction to be h V instead of SV. In the Figure

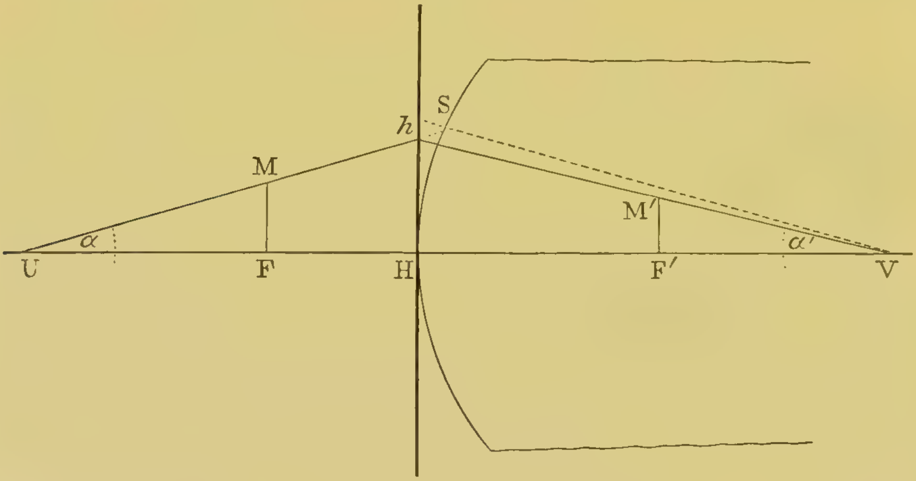

Fig. 128.

$$UH = u,\ FH = f$$
$$HV = v,\ HF' = f'$$

Erecting perpendiculars from F and F′ to meet the incident and refracted ray, and putting $FM = z$, $F'M' = z'$, and $Hh = y$, we have—

$$\frac{z}{y} = \frac{u-f}{u} \text{ and } \frac{z'}{y} = \frac{v-f'}{v} \text{ and as from 3).}$$

$$\frac{u-f}{u} + \frac{v-f'}{v} = 1$$

$$\frac{z}{y} + \frac{z'}{y} = 1 \text{ or } z + z' = y.$$

This proves Neuman's theorem, that the distance from the axis of the point at which an incident and refracted ray meet the tangent plane to the vertex is equal to the sum of the perpendiculars drawn from the foci to meet the two rays. If U lie between F and H, both y and z' have opposite signs from z.

By the aid of this theorem we may draw the direction of any ray after refraction at one surface, as, even although it should not emanate from a point on the axis, it will have such a direction (prolonged if necessary) as to cut the axis at some point, and may be looked upon as proceeding from that point. It may easily be shown that rays proceeding from any point outside the axis meet after refraction at another point, when the required relation between the perpendiculars from the foci exists. In the case of one surface, however, we may look upon every ray which is directed towards the centre of the surface as an axial ray, so that by the simple construction shown in Fig. 129 we can easily find a point conjugate to any other.

Let P be the point whose conjugate focus is to be found, XX′ the axis, and QHR the tangent plane, to the refracting surface of which C is the centre, F and F′ the foci. From P draw PC through the centre. This ray being directed towards the centre passes unrefracted. Draw PQ parallel to the axis XX′. This must pass after refraction through F′, as, being parallel with the axis, it is brought to the principal focus. Now as the focus of P must lie on both these lines, PC and QF′, it must be where they meet or at P′. The same point would be got by drawing from P a ray passing through the anterior principal focus, which on refraction must proceed parallel to the axis XX′.

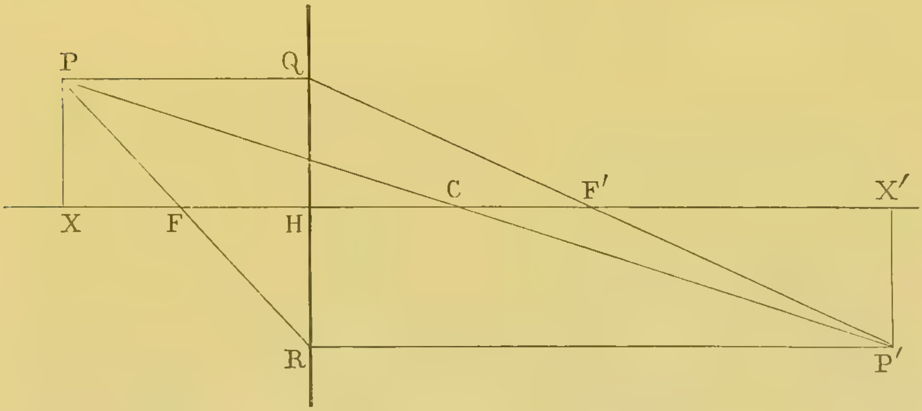

Fig. 129.

Now let the points on which perpendiculars from P and P′ meet the axis be X and X′. Then HQ = XP and HR = X′P′:

$$\frac{XP}{X'P'} = \frac{HX - f}{f}$$

$$\frac{X'P'}{XP} = \frac{HX' - f'}{f'}$$

From which,

$$(\mathrm{HX}-f)\ (\mathrm{HX}'-f')=ff',$$

i.e., X and X′ are conjugate foci, or *the abscissæ of conjugate points outside the axis are also conjugate.* Any point on XP has an image on X′P′, and consequently X′P′ is the image of XP, that is, to the required degree of approximation the image of a straight line is also a straight line, and it may be shown that to the same approximation the image of a small portion of a plane is also a small portion of another plane. In reality an object, any point of which lies on a sphere which is concentric with the spherical refracting surface, has its most distinct image on the surface of another concentric sphere. When, however, only very small portions of these spherical surfaces are considered, they may be looked upon as coincident with their tangent planes.

From Fig. 129 we see that the relation between object and image for refraction at one spherical surface, putting $\mathrm{HX}=u$ and $\mathrm{HX}'=v$ and $\mathrm{HC}=\mathrm{R}$, is :

$$\frac{obj}{im}=\frac{u+\mathrm{R}}{v-\mathrm{R}}\quad . \quad . \quad . \quad . \quad 6.)$$

The tangent plane at the vertex, a section of which is represented in the figure, is called the *principal plane*, and the point H, where it meets the axis, the *principal point.* An object in the principal plane has its image also in the same plane, *i.e.*, *object and image coincide in the principal plane*, for if we put $u=o$, the equation for conjugate points or images $(u-f)\ (v-f')=ff'$, is only satisfied by $v=o$.

Further, *object and image in the principal plane are of the same size.* This is seen by putting $u=o$ and $v=o$ in 6).

The centre of curvature through which rays joining conjugate points pass directly, is called the *nodal point.* Rays which are directed towards the nodal point before refraction pass after refraction as if they came from that point. The nodal point is evidently its own image.

The principal point, the nodal point, and the two principal foci are known as the *cardinal points.*

Where instead of only one refracting surface we have to do with a number of such surfaces, separated by different refractive media, and having a common axis, a great simplification in the calculation of the relation of object and image is made by determining the position of the cardinal points, of which in general there are three pairs, viz., two principal points, two nodal points, and two principal foci. The formulæ for the calculation of these cardinal points are lengthy but symmetrical, so that it is easy to deduce from a system of n surfaces those for a system of $n+1$ surfaces.

In problems connected with errors of refraction of the eye, we have more to do with the alteration which is effected in the position of the cardinal points when glasses of different strengths, and placed at different distances in front of the eyes, are used, than with the calculation of their actual position.

A number of centred surfaces, $S_1S_2 \ . \ . \ . \ S_n$, are represented in Fig. 130 by the tangent planes to their vertices, which we have seen may be done in the case of one surface, and may therefore (as the image formed by the first surface may be looked upon as the object of the second, and so on) be done for all. Take a ray BQ, meeting the first surface parallel to the axis, AA′, and one, Q′B′, on the same level, also parallel to the axis after passing the last surface; BQ will, after refraction through all the different media, pass through F′, the second principal focus of the system, and Q′B′ will be the direction of a ray which passes through the point F, the first principal focus of the system. Let LF′ be the direction of BQ after refraction, and FR that of QB′ before refraction, FR and BQ prolonged must meet at some point h, so must also B′Q′ and F′L, say at h', a point at the same distance from the axis as h. It follows from the construction that there will be in general two points related to each other as h and h'. Now it is evident that rays which meet on the one point proceed as if they came from the other, and therefore the one point is the image of the other. They are conjugate points, and consequently the planes perpendicular to the axis on which they lie are also conjugate. hH and h'H′ are sections of those planes, and are called the *principal planes* of the system of surfaces; hH having reference to the rays coming from the first medium, is called the *first principal plane*, and h'H′ having reference to the direction of corresponding rays in the last medium, the *second principal plane*. They possess the same property as the one principal plane in the case of a single refracting surface, viz., that object and image are the same size. (In the Fig. hH is the image of h'H′, and h'H′ is the image of hH, and further, $h\text{H} = h'\text{H}'$.) The points where these planes cut the axis H, H′ are the principal points, and as in the case of one surface the focal distances were measured from the principal point, so for any system of centred surfaces they are measured from the two principal points—the first

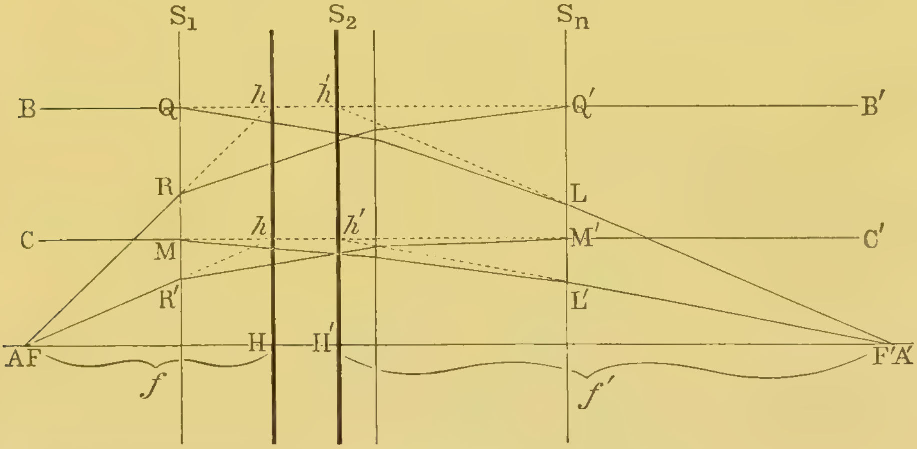

Fig. 130.

from the first, and the second from the second. In the figure the actual course of two rays from either focus is represented in the case of three surfaces, from which it is seen that their direction after refraction is given by prolonging them till they meet the first principal plane, and then drawing them parallel to the axis, as if they came from points in the second principal plane, equidistant from the axis with the points to which they are directed on the first. When we know the position of the principal planes for any system of surfaces, and the focal distances measured from them, we may easily find the position of a point conjugate to any other by a construction which is similar to that of Fig. 129.

From the point P (Fig. 131), draw PQ parallel with the axis XX′ and PR, passing through the anterior principal focus F. PQ, after refraction, passes then through F′, and as if it came from Q′. PR again proceeds from R′ parallel with the axis; the image of P must lie on both these rays, and therefore at P′, where they meet. From the figure

$$\frac{\mathrm{FH}}{\mathrm{PQ}}=\frac{\mathrm{RH}}{\mathrm{RQ}}, \qquad \frac{\mathrm{F'H'}}{\mathrm{P'R'}}=\frac{\mathrm{Q'H'}}{\mathrm{Q'R'}}$$

putting f and f' for FH and F′H′, and u and v for PQ and P′R′, and remembering that RQ = R′Q′, we get by addition $\frac{f}{u}+\frac{f'}{v}=1$, or the same formula as for a single surface. From P and P′ drop perpendiculars on the axis; P′X′ is then the image of PX, and from the figure we see that as RH = P′X′

$$\frac{\mathrm{PX}}{\mathrm{P'X'}}=\frac{u-f}{f}=\frac{f'}{v-f'}$$

or adding numerators and divisors together

$$\frac{obj}{Im}=\frac{u+(f'-f)}{v-(f'-f)} \quad . \quad . \quad . \quad . \quad 6a.)$$

This is very similar to 6), $f'-f$ standing for R, and if we measure along the axis, from the first and second principal points respectively, two lines equal to $f'-f$, we get the two *nodal points* N and N′, which correspond to the one nodal point of a single surface. These are evidently separated by the same distance which separates the two principal points. The one nodal point, too, is the image of the other, for the equation for conjugate points is satisfied when for u we put $-\mathrm{N}=-(f'-f)$ and for v, $\mathrm{N'}=(f'-f)$, *i.e.*—

$$\frac{f}{-\mathrm{N}}+\frac{f'}{\mathrm{N'}}=1$$

In Fig. 131 we see that as from 6*a*.) $\frac{\mathrm{XN}}{\mathrm{X'N'}}=\frac{\mathrm{PX}}{\mathrm{P'X'}}$ and XN is in the same line with X′N′, and PX is parallel with P′X′, the triangles PXN and P′X′N′ are similar, and P and P′ are any pair of conjugate points. The nodal points are so situated then that *rays which before refraction are directed towards the first, appear after refraction as if they came from the second, and have a direction parallel to their original one.*

There is an important relation between the focal distances for any system of refracting surfaces which have a common axis, viz., *whatever be the number of refracting media, the focal distances (measured from the principal points) are to each other as the index of refraction of the first medium is to that of the last.* This theorem may be proved in a number of ways. The following proof is based on the relation existing between the angles of deviation of an incident and refracted ray.

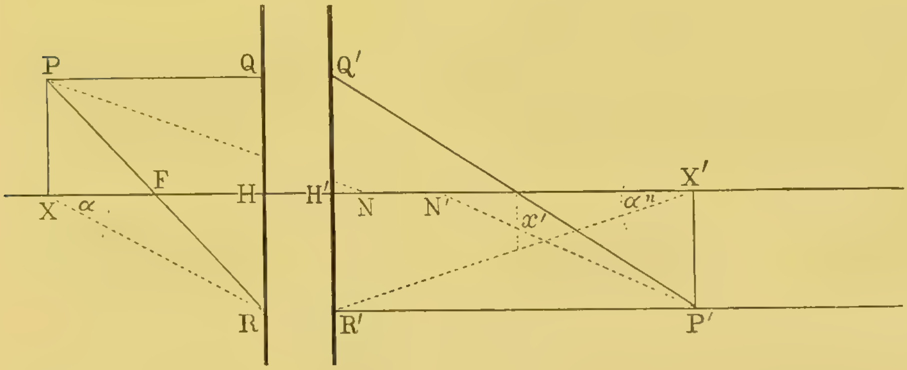

FIG. 131.

In Fig. 128 we have $Hh = u \tan \alpha = -v \tan \alpha'$ and from 1 *b*)

$$v\,(u + R) = \mu u\,(v - R)$$

$$\therefore \frac{u + R}{v - R} = \frac{\mu u}{v}$$

$$= \frac{\mu \tan \alpha'}{\tan \alpha}$$

i.e.,—

$$\frac{obj}{im} = \frac{\mu \tan \alpha'}{\tan \alpha}$$

Now if there are *n* media, and we call the index of refraction of the first μ_0 and of the last μ_n and the size of the first object O, that of its successive images $O_1 O_2 \ldots O_n$ we should have

$$\begin{aligned} \mu O \tan \alpha &= \mu_1 O_1 \tan \alpha_1 \\ &= \mu_2 O_2 \tan \alpha_2 \\ &\quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \\ &= \mu_n O_n \tan \alpha_n \end{aligned}$$

(Helmholtz, *Phys. Opt.*, page 75.)

Again, from Fig. 131 we see that :—

$$O : O_n = f' : X'F'$$

$$\therefore \mu_o f' \tan \alpha = \mu_n X'F' \tan \alpha_n$$

$$= \mu_n z'$$

When X moves up to F, $z' = HR = f \tan \alpha$, in which case

$$\mu_o f' \tan \alpha = \mu_n f \tan \alpha$$

or

$$\frac{\mu_o}{\mu_n} = \frac{f}{f'} .$$

In the case of a glass or other lens in air, the two focal distances are the same, and consequently the nodal points coincide with the principal points. For the lenses (biconvex and biconcave) in general use, the principal planes lie within the lens, so that the interval separating them is less than the thickness of the lens. They may consequently be looked upon for most practical purposes as coinciding with one vertical line which divides the lens into two similar halves, the nodal points being also merged into one at the point where the line cuts the axis.—See Fig. 132.

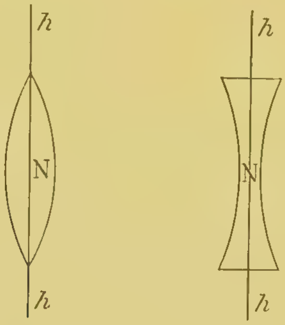

Fig. 132.

There is a very considerable difference in the position of the cardinal points in different eyes. They also differ, of course, according to the state of accommodation of the eye. Helmholtz's most recent measurements and calculations give the following average values for the eye at rest—anterior focal distance (f) 15·498 millimetres, posterior focal distance (f') 20·713 millimetres. First principal point (H) = 1·753 millimetres behind surface of cornea—second principal point (H′) = 2·106 millimetres behind surface of cornea—first nodal point (N) = 6·968 millimetres, and second nodal point (N′) = 7·321 millimetres behind surface of cornea. On the assumption of a full accommodative change, by which the radius of curvature of the anterior surface of the lens is altered from 10 millimetres to 6 millimetres, that of the posterior surface from 6 millimetres to 5·6, and the thickness of the lens increased from 3·6 to 4 millimetres, so that its anterior surface is approximated by 0·4 millimetres to the cornea, the respective values of those quantities are changed to the following:—

$f = 13{\cdot}99$ millimetres, $f' = 18{\cdot}692$ millimetres, H from cornea = 1·858 millimetres, H′ from cornea = 2·257 millimetres, N from cornea = 6·566 millimetres, N′ from cornea = 6·965 millimetres.

From these figures it will be seen that the interval between the first and second principal points and between the first and second nodal points is very small, 0·353 millimetres for the unaccommodated, and 0·399 millimetres for the accommodated eye. The average radius of curvature of the cornea was taken at 7·829, so that in the eye at rest the second nodal point (the most important on account of the influence of its position on the size of the retinal images) lies very nearly $\frac{1}{2}$ millimetre in front of the centre of curvature of the cornea, whilst in the accommodated eye it advances so as almost to coincide with the first nodal point of the eye at rest.

When glasses are placed in front of the eye, we have to do with a combination of two systems of centered refracting surfaces, and must know what is the difference in the position of the cardinal points of the eye alone and those of the system composed of eye and lens, in order to understand rightly their effect on the sizes of the images formed on the retina.

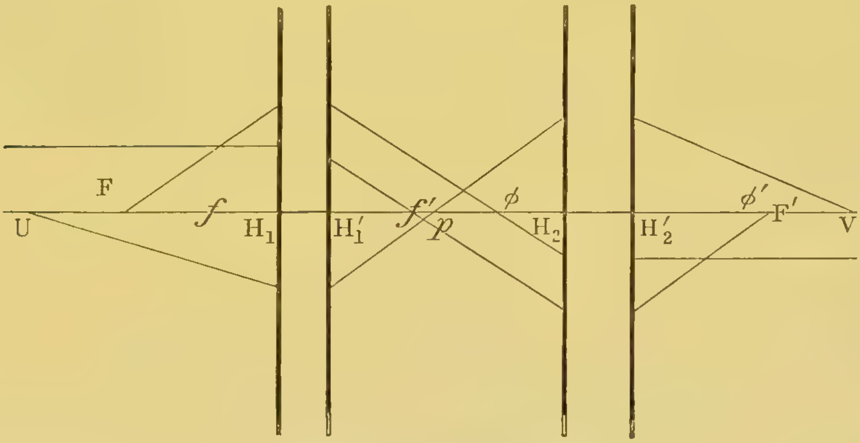

FIG. 133.—(From Helmholtz.)

Helmholtz has given the following simple method of combining two systems whose cardinal points and the distance intervening between any two cardinal points in the two systems are known. In Fig. 133, H_1 H'_1 are the principal points of the first system and H_2 and H'_2 those of the second system. Let the distance intervening between the second principal point of the first system and the first principal point of the second system be denoted by D, *i.e.*,

$$H'_1 H_2 = D,$$

f and f' are the focal distances of the first, ϕ and ϕ' those of the second system, F and F′ those again of the combined systems. The anterior focus of the combined systems is evidently the image on refraction through the first system of rays proceeding from the anterior focus of the second system. Putting then

$$D-\phi \text{ for } v, \text{ in the equation } \frac{f}{u}+\frac{f}{v}=1$$

we get

$$H_1F=\frac{(D-\phi)\,f}{D-\phi-f'}$$

Similarly

$$H'_2F'=\frac{(D-f')\,\phi'}{(D-f')-\phi}.$$

The first condition for the principal points is that they should be in the relation of object to image. They must consequently be the images given by each system of some other point. Let P be that point and U and V its images after refraction through the first and second systems respectively. In order that U and V shall be the principal points, they must satisfy the further condition that images in the perpendicular planes, through these points shall be equal in size and similarly directed. Putting S for the image at P, O and O′ for the images at U and V, $x=H'_1P$ and $y=H_2P$
we have

$$\frac{O}{S}=\frac{f'}{f'-x}$$

and

$$\frac{O'}{S}=\frac{\phi}{\phi-y}.$$

To have $O=O'$ we must have

$$\frac{f'}{f'-x}=\frac{\phi}{\phi-y}$$

or

$$\frac{x}{f'}=\frac{y}{\phi}$$

as $x+y=D$, we have

$$\frac{x}{f'}=\frac{D-x}{\phi} \text{ and } \frac{y}{\phi}=\frac{D-y}{f'}$$

$$\therefore x=\frac{Df'}{\phi+f'}, \text{ and } y=\frac{D\phi}{\phi+f'}.$$

Again

$$H_1U=\frac{xf}{x-f'} \text{ and}$$

$$H'_2V-\frac{y\phi'}{y-\phi}$$

and putting in the above values for x and y, these distances H_1U (which we may call h), and H'_2V (which we may call h'), which are the distances respectively of the first principal plane of the combined system from the first principal plane of the first system, and of the second principal plane of the combined system from the second principal plane of the second system, have the following values:—

$$\left.\begin{aligned} h &= \frac{\mathrm{D}f}{\mathrm{D}-\phi-f'} \\ h' &= \frac{\mathrm{D}\phi'}{\mathrm{D}-\phi-f'} \end{aligned}\right\} \quad . \ . \ . \ . \ . \ . \quad 7.)$$

For F and F′ we have

$$\left.\begin{aligned} \mathrm{F} &= \mathrm{H}_1\mathrm{F} - h = \frac{\phi f}{\phi+f'-\mathrm{D}} \\ \mathrm{F}' &= \mathrm{H}'_2\mathrm{F}' - h' = \frac{\phi' f'}{\phi+f'-\mathrm{D}} \end{aligned}\right\} \quad . \ . \quad 8.)$$

In the problems connected with the eye, the first system is a lens in front of the eye surrounded by air, and therefore $f=f'$. In this case the values of hh', F and F′ are further simplified, and we have

$$\left.\begin{aligned} h &= \frac{\mathrm{D}f}{\mathrm{D}-\phi-f} \\ h' &= \frac{\mathrm{D}\phi'}{\mathrm{D}-\phi-f} \\ \mathrm{F} &= \frac{\phi f}{\phi+f-\mathrm{D}} \\ \mathrm{F}' &= \frac{\phi' f}{\phi+f-\mathrm{D}} \end{aligned}\right\} \quad . \ . \ . \ . \ . \quad 8a.)$$

If the lens be placed in front of the eye at such a distance from its first principal plane as to make $\mathrm{D}=\phi$, then

$$\begin{gathered} h = -\phi \\ h' = -\frac{\phi\phi'}{f} \\ \mathrm{F}=\phi \text{ and } \mathrm{F}'=\phi'. \end{gathered}$$

The first principal plane remains then unaltered in position, whilst the second is moved to an extent depending on one variable only, viz., the focus of the lens in front of the eye, and backwards or forwards, according as that is negative or positive. As the distance of the first nodal point from the first principal plane, and of the second from the second principal plane is equal to the difference between the two focal lengths, it follows that when $\mathrm{D}=\phi$ the first nodal point, too, remains unaltered in position, and the second is moved to the same extent as the second principal plane.

Where the ametropia is axial (which, as was first shown by Donders, is most frequently the case) the degree of elongation or shortening of the antero-posterior axis of the eye is equal in extent to the alteration in the position of the second nodal point if the correcting lens be placed at the anterior focal point of the eye.

This theorem is proved in the following way: If ϕ and ϕ' be the anterior and posterior focal distances of the eye, we may express the relation between the conjugate foci u and v, which represent respectively the distance of an object from the first principal plane and its image on the retina, and therefore the distance of the retina itself from the second principal plane of the eye, by the formula 5 β.

$$v - \phi' = \frac{\phi\phi'}{u - \phi} \cdot$$

Now when the lens correcting the ametropia is at the anterior focal point of the eye, its focal distance f may be put for $u - \phi$ in the above equation, which then becomes

$$v - \phi = \frac{\phi\phi'}{f} \cdot$$

The distance from the second principal focus of the eye to the retina varies therefore indirectly as f. If we take f at 1 metre, we have as the axial elongation or shortening, producing a myopia or hypermetropia of the extent corrected by a minus or plus lens of 1 dioptre (taking Helmholtz's values of ϕ and ϕ'),

$$= \frac{15{\cdot}498 \times 20{\cdot}713}{1000} = 0{\cdot}32 \text{ mm.}$$

From this we see what a comparatively trifling change in the length of the eye is sufficient to give rise to a considerable degree of ametropia. A deviation in either direction, for instance, to the extent of the breadth of a shilling would require, for the correction of the abnormality in refraction which it would produce, a lens of rather more than 3 dioptres if placed about 14 millimetres from the cornea.

When an axial ametropia is corrected by a lens placed at the anterior focus of the eye, the retinal images which it receives are the same size as if the eye were emmetropic.

This is evident from the fact that the second nodal point of the combined system of lens and eye lies just as far in front of or behind that of the eye, as the ametropic retina lies in front of or behind the position it should occupy to coincide with the principal focus of the dioptric media of the eye.

Numerous measurements of the curvature of the cornea in cases of ametropia have shown that although the highest degrees of myopia and hypermetropia are often associated with respectively a smaller and greater radius of curvature of the cornea, still there appears as a rule rather to be a tendency on the part of the corneal curvature towards a correction of the axial abnormality.

When the correcting lens is placed at a greater distance than on the average 13-14 millimetres in front of the eye, the corresponding axial elongation is less, and the corresponding axial shortening greater than when placed in that position (*i.e.*, the anterior focal point of the eye). Conversely, when the correcting lens is placed at a shorter distance from the eye than 13-14 millimetres, the corresponding axial elongation is greater and the shortening less than when it occupies that position. In other words—

An axial myopia requires a stronger lens for its correction the further the lens is placed from the eye; an axial hypermetropia, on the other hand, a weaker one.

To see that this is the case, we have only to make D greater or less than ϕ, when we get $u-\phi$ greater than f for a positive glass, between the eye and its anterior focus, and for a negative glass farther from the eye than its anterior focus; whilst $u-\phi$, less than f, would stand for a positive glass farther from the eye than its anterior focus, and for a negative glass between the eye and that point. If x be put for the difference between f and $u-\phi$, we have in the first case—

$$v-\phi'=\frac{\phi\phi'}{f+x}, \text{ and for the second}$$

$$v-\phi'=\frac{\phi\phi'}{f-x}$$

In order to see what change takes place in the positions of the cardinal points when the interval between the glass and the eye is increased more and more from $D=\phi$, we may, as above, put $D=\phi+x$, x standing for the variable increase. Putting this value for D in the expression for F and F′ in 8*a*), we get—

$$F=\frac{\phi f}{f-x} \qquad \text{and}$$

$$F'=\frac{\phi' f}{f-x}$$

$$\therefore \quad \frac{F}{\phi}=\frac{F'}{\phi'}=\frac{f}{f-x}$$

$$\text{and} \quad \frac{F}{F'}=\frac{\phi}{\phi'}.$$

That is, the focal distances of the combined system are greater when a convex glass is further from the eye than its anterior focus, or a concave nearer than its anterior focus, and less when a concave lens is further from, and a convex lens nearer, the eye than that distance.

Also the distance of the nodal points $=(F'-F)$, from the principal points is greater or less under the same circumstances. Again, putting the same value for D in the expression for h and h' in 8*a* we get—

$$h=\frac{(\phi+x)f}{x-f} \quad \text{and}$$

$$h'=\frac{(\phi+x)\phi'}{x-f}.$$

Now when f is negative the above value for h increases with x, though not so rapidly as x, consequently the first principal point advances as a concave lens is withdrawn from the eye. When f is positive, the value for h is also increased, and more rapidly than x; so that the first principal point recedes more and more the further a convex lens is withdrawn from the eye. Again, when f is negative, h' is positive, and increases with x; therefore, the further a concave glass is in front of the eye, the more does the second principal point of the system recede from the position occupied by the same point in the eye. When f is positive h' is negative, and increases with x; therefore, the further a convex glass is held from the eye the more does the second principal point of the system of eye and lens advance from that occupied by the same point for the eye alone. As the distance from the first principal point to the first nodal point $=F'-F$, and this in the case of a concave glass, further from the eye than its anterior focal distance, is less than $\phi'-\phi$, it follows that the first nodal point also advances more and more and to a greater extent than the first principal point, when a concave lens is removed from the eye. The second nodal point, which lies at the same distance $F'-F$ from the second principal point, recedes more and more, though to a less extent, than the second principal point as a concave lens is withdrawn from the eye. This is evident, as although $F'-F$ is less than $\phi'-\phi$, its value, $\frac{(\phi'-\phi)f}{f-x}$ when f is negative, does not diminish so rapidly with the increase of x, as the value of h', viz., $\frac{(\phi+x)\phi'}{x-f}$.

Again, when f is positive, $F'-F$ is greater than $\phi'-\phi$ when the convex lens is further from the eye than its anterior focal point; therefore, as this position of the lens causes the anterior principal point to recede, it follows that the anterior nodal point recedes still further. The second nodal point advances, though not so far as the second principal point, as there is the same distance between the two nodal points as between the two principal points. This is evident, too, from the above expression for h' increasing more rapidly with x than does $F'-F$.

When the ametropia is corrected by a glass situated further in front of the eye than its anterior focal point, the image received on the retina of the eye at rest is smaller in the case of myopia, *and greater in the case of hypermetropia, than that which would be received by an emmetropic eye under the same conditions without a glass. The opposite is the case if the correcting glass is placed nearer the eye than its anterior focal point.*

This may be shown as follows:—The distance from the second nodal point N′ to the second principal focus F′ (the retina in the case of corrected ametropia) $= F' - (F' - F) = F = \frac{\phi . f}{f - x}$. As the focal distance (f) of the glass to correct the ametropia increases or decreases to the same amount as x, the denominator of this expression for F always remains the same whilst the numerator v varies with f. Now we have seen (1.) that the correcting glass for myopia is stronger and therefore f less, the correcting glass for hypermetropia, on the other hand, weaker, and therefore f greater the further it is from the eye; (2.) that when the correcting glass is at the anterior focal point of the eye the distance between the second nodal point and the retina is the same as in the case of emmetropia; F, however, becomes smaller with f, that is, with the increase in strength of the dioptre lens used for correction; therefore the distance from the second nodal point to the retina becomes less the stronger the correcting lens, and it is less for correction with a concave glass in front of the anterior focus of the eye, and for a convex one between the eye and its anterior focus than in an emmetropic eye of equal refracting power. F, on the other hand, increases with f, that is, with the diminution in the strength of the correcting lens; therefore the distance between the second nodal point and the retina is greater the weaker the correcting lens, and is greater for correction with a concave glass nearer the eye than its anterior focal point, and with a convex lens placed beyond that point, than in the case of a corresponding emmetropia. But we have seen that the sizes of the retinal images are larger the further the correcting convex glass is from the eye, or the nearer the correcting concave glass, and *vice versa*. As an example, take the correction of an ametropia by a lens of 12 D. This will correspond for correction at anterior focus to an axial change of 3·84 millimetres, and in the case of myopia will also be corrected by a −13·0 D lens, 6·4 millimetres in front of, or a −11·0 D lens, 7·6 millimetres behind the anterior focal point of the eye. In the case of hypermetropia, again, the correction is also effected by +11·0 D lens, 7·6 millimetres in front of, and a +13·0 D lens, 6·4 millimetres behind the anterior of the eye. In both cases we have for correction by a 12·0 D lens, $F = 15{\cdot}5$; by a 13·0 D, $F = \frac{15{\cdot}5 \times 76{\cdot}9}{83{\cdot}3} = 14{\cdot}31$; by an 11·0 D $F = \frac{15{\cdot}5 \times 90{\cdot}9}{83{\cdot}3} = 16{\cdot}91$, the relative sizes of the images being—

for correction with 12·0—1.
" " 13·0—0·9168.
" " 11·0—1·091.

The position of the crystalline lens in the eye has an influence on the refraction of the eye. Thus, if there has been previously emmetropia, an advance of the lens produces myopia, a recession, hypermetropia.

To see this we have only to notice what must be the effect of increasing D, put for the interval between the apex of the cornea and the first principal point of the lens in the formulæ for the values of h' and F′. As D increases both F′ and h' increase, but F′ more rapidly than h'; consequently the second focal distance of the eye increases with D. If for one value of D (usually about 6 millimetres) the eye is emmetropic for a greater value, the retina lies in front of the focus, or the eye is hypermetropic, while for a less value it lies behind the focus, and the eye is consequently myopic. A change of less than $\frac{1}{2}$ millimetre in the position of the lens is sufficient to cause an ametropia of 1 D.

We have seen that spherical lenses have the effect of altering the position of the range of accommodation. They also produce a change in the actual effective amount of that range. Concave glasses increase and convex diminish the effective range of accommodation. Myopes have therefore a certain advantage over others in respect of accommodation, as not only have they a closer near point without correction, and a suitable far point with correction, but their effective range of accommodation is increased by the glass which they wear for the improvement of their distant vision. Hypermetropic individuals, on the other hand, have the double disadvantage of a frequently too-distant near point without correction, and a curtailment of the range of accommodation with correction.

The influence of spherical correcting lenses on the accommodative range (A) may be shown in the following way:—The addition to the refractive power, which takes place by the alteration of the curvature of the lens, may, as Nagel has shown, be most conveniently looked upon as producing the same effect as a thin lens of the power A situated at the first principal point of the eye. We have seen that $N - F = A$, *i.e.*, the difference between the dioptric value of the lens, which at the first principal point of the eye has its focus equal to the distance of the near point (measured from the principal point), and the dioptric value of that which in the same situation corresponds in focal value to the distance of the far point, is equal to the accommodative change. This difference, therefore, represents the range of accommodation. Now let us imagine the lens correcting an ametropia to be situated also in the first principal plane; it is obvious that the far point before and after correction will be conjugate points with respect to this imagined lens. If we call the dioptric value of the first F, and of the second F′, and the value of the lens L, we have therefore

$$F' - F = L \quad . \quad . \quad . \quad a)$$

Inasmuch, however, as it is of course impossible to give the correcting lens this position, we may call the value of the correcting lens in front of the eye L′. (This, we have seen, is greater in myopia and less in hypermetropia.) The distances of the near point from this lens before and after correction are conjugate points with respect to it. If we call these, as represented by their dioptric values, X and X′, we have

$$X' - X = L' \quad . \quad . \quad . \quad \beta)$$

subtracting α from β we get

$$(X' - F') = (X - F) + (L' - L).$$

Now putting N and N′ for the distances of the near point from the principal point of the eye before and after refraction through L′ :—

$$(X' - F') = (N' - F') + (X' - N') = A' + (X' - N')$$

and

$$(X - F) = (N - F) + (X - N) = A + (X - N)$$

$$\therefore A' = A + (L' - L) + (X - X') + (N' - N) \text{ and}$$

$$X - X' = -L'.$$

When L fully corrects the ametropia—

$$L = F \therefore A' = N'.$$

For the correction of myopia L is negative and (N′ − N) negative, but less than L. Consequently in myopia A′ is greater than A. For the correction of hypermetropia L and N′ − N are positive, but L greater than (N′ − N). Consequently in hypermetropia A′ is less than A. As N′ − N does not increase so rapidly as L, the increase or diminution in the range of accommodation increases with the strength of the lens used. As examples take the cases of 5 D of myopia, or hypermetropia with 10 D range of accommodation, and fully corrected by a lens 15 millimetres in front of the first principal point of the eye. In the case of M = 5·0 D, N = 15·0 D and X 19·38 D ∴ as L′ = 5·405 D, X′ = 13·975 D, and A′ = N′ = 11·553 or a gain of 1·5 D. For H 5·0 A′ = N′ = 8·738 or a loss of 1·27 D.

Again, take the case of a hypermetropia 6·0 D, and with a range of accommodation of 8·0 D, partially corrected by a + 3·0 D lens, 20 millimetres in front of the first principal plane.

Here L = 3·19 D and A′ = 8 + 4·62 − 2·0 − 3·19 = 7·43 D, or a loss of 0·57 D.

ABSOLUTE AND RELATIVE VISUAL ACUITY.

We have seen (p. 524) that where an axial ametropia is corrected by a lens whose second principal plane coincides with the anterior focal plane of the eye, the retinal image of a distant object is the same size as if the eye were emmetropic.

In Fig. 134, A and B are two rays passing from two extremities of a distant object. A passes through ϕ, the anterior focus of the eye, and after meeting the first principal plane of the eye proceeds parallel with the axis. B coincides with the axis itself, $\alpha\ \beta$ consequently represents the size of the retinal image of the distant object. That the retinal image $\alpha\ \beta$ will be the same size, whether the eye be emmetropic and therefore focussed for the distant object without any accommodation, or ametropic and corrected by a lens whose second principal plane passes through ϕ, is seen in Fig. 135 as A and B suffer no deflection on passing through the lens L L, whose optical centre coincides with ϕ.

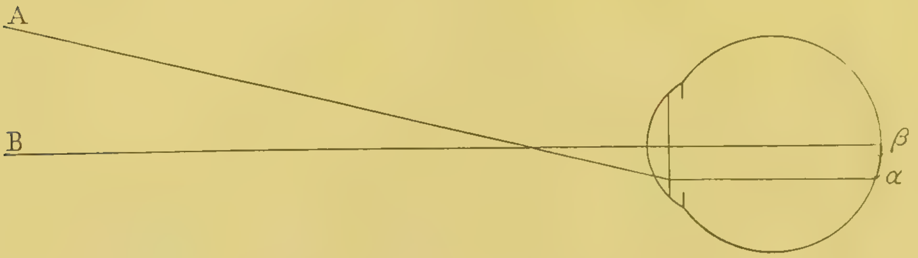

FIG. 134.

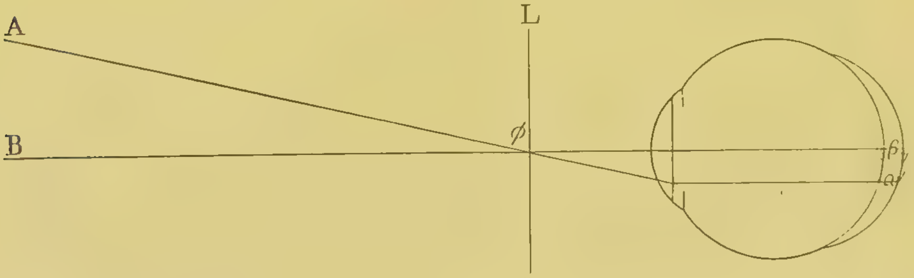

FIG. 135.

Further, the same retinal image will correspond to the same angle A ϕ B, whatever be the distance of the object accurately focussed in the eye, provided the dioptric arrangement of the eye is similar in all cases and does not alter by accommodation. Thus the retinal image

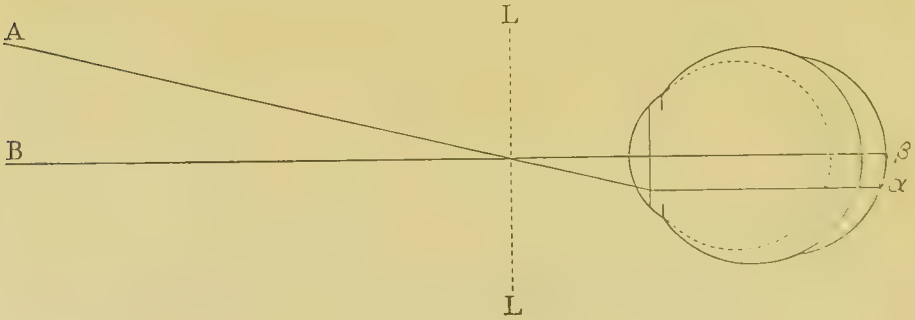

FIG. 136.

of an object situated at the far point of an eye with axial myopia will be the same size as the image in an emmetropic eye at rest and focussed by a convex lens which corrects for the same distance, and the same size again as the image of the same object on the retina of an axial hypermetropic eye corrected by a stronger convex lens for the same distance. This is seen in Fig. 136.

Now, if we take the retinal image $\alpha\,\beta$ so small that it can just be distinguished, that is to say (see Chap. I.), if $\alpha\,\beta$ be the smallest distance separating the retinal images of two objects which it is just possible to recognise as discrete, we have the measure of the *absolute visual acuity.*

As $\alpha\,\beta = \frac{AB \cdot H\phi}{B\phi}$ the absolute visual acuity is got when AB is the smallest discernible body at the distance $B\phi$ from the anterior focus, for which the unaccommodated eye is focussed with or without a correcting lens at its anterior focus.

To compare the visual acuity on accommodation, or the *relative visual acuity* with the absolute, we have merely to compare the size of the object corresponding to the smallest retinal image on accommodation with that at the same distance from the unaccommodated eye, focussed instead by a lens situated at its anterior focus.

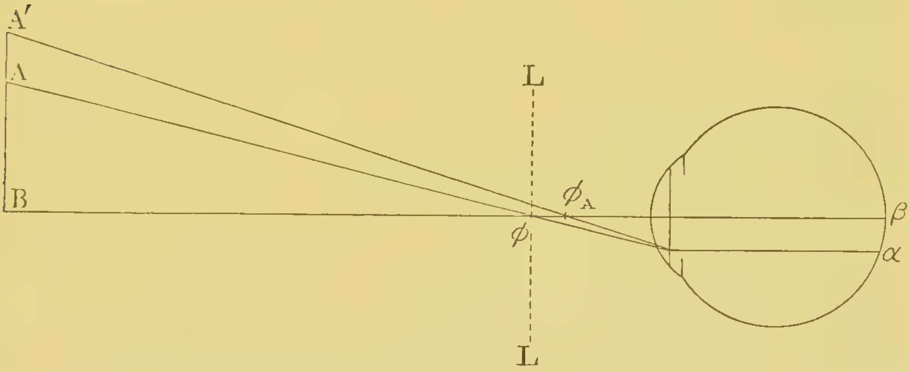

FIG. 137.

As the anterior focus of the accommodated eye is shorter, the nature of the difference is shown in Fig. 137, where ϕ_A represents the altered position of the anterior focus.

From the figure it is seen that the image $\alpha\,\beta$ thus corresponds to a larger object, and as this holds good at the limit, the relative visual acuity is less than the absolute.

We may, without introducing an error of any consequence, look upon the accommodation of the eye as effected by a thin lens (of focus $f = u$, the distance for which accommodation takes place). For the new anterior focal distance of the altered eye we have from 8*a*.) $F = \frac{\phi u}{\phi + u}$. As no change takes place in the position of the first principal point, this formula is sufficient to determine that of ϕ_A.

Now, as the focal distance of the eye is small compared with the distance for which accommodation ordinarily takes place, we may with sufficient accuracy look upon the sizes of the retinal images of an object accommodated for, or obtained by, a lens at the anterior focus of the eye at rest as proportional to the respective distances of the anterior focus (see Fig. 138). In the limit, therefore, we have relative visual acuity is to absolute visual acuity as altered anterior focus is to anterior focus of eye at rest, *i.e.*,

as $$\frac{\phi u}{\phi + u} : \phi \quad . \quad . \quad . \quad . \quad . \quad 9.)$$

Put a for $\frac{1}{u}$, which, if u be reckoned in metres, represents the dioptre lens of focus u 9), then becomes—

$$\frac{1}{1 + a\phi} \quad . \quad . \quad . \quad . \quad . \quad 9a.)$$

The average value for ϕ, as given by Helmholtz, is ·015498 metre.

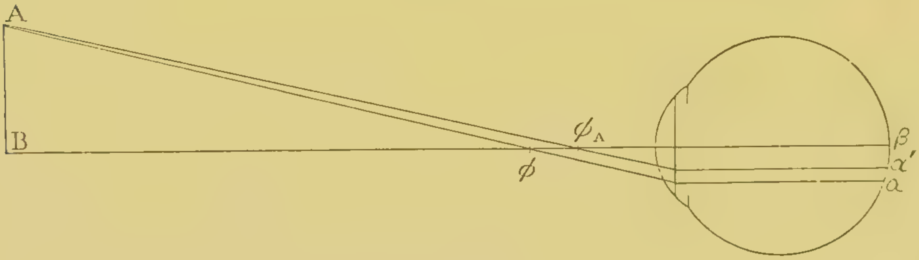

FIG. 138.

We have, therefore, as a formula for calculating the value of the relative visual acuity or a dioptre of accommodation—

$$\frac{1}{1 + \cdot 015498a} \quad . \quad . \quad . \quad . \quad . \quad 9\beta.)$$

For 4 dioptres of accommodation, or about what is required for focussing the eye for the reading distance, the relative visual acuity (if the absolute acuity be taken as unity) would be ·9416, a difference of about 6 per cent. only, or 1½ per cent. for every dioptre of accommodation. This amount, though hardly of any practical import, is sufficient to make a quite appreciable difference in the apparent size. We can, for instance, readily distinguish an equality in the length of objects differing much less than 6 per cent. from each other.

Gullstrand has by a different and somewhat lengthy analysis arrived at the same result as to the relative visual acuity as I have here given. He has pointed out that the ordinary way of deducing the change in the visual acuity on accommodation from the degree of displacement of the second nodal point is wrong.

From the above it follows that the visual acuity of an uncorrected hypermetrope tested at a distance in the ordinary manner is less by 1½ per cent. for every dioptre of hypermetropia than his absolute visual acuity.

In aphakia, too, the absolute visual acuity may be taken as the acuity found when correction is made at the anterior focus of the eye. This is greater than in the normal eye in the proportion of the aphakic anterior focal distance to the focal distance of the emmetropic eye of the ordinary build, or the ametropic eye of the same optical build corrected at its anterior focal point. The proportion is something like 23 : 15, or rather more than 3 : 2. Just as in hypermetropia the acuity is increased the further the correcting lens lies in front of the cornea.

HYPERMETROPIA.

This is the most common condition of refraction, and is more common at an early age than in adult life. The eyes of new-born infants are almost invariably hypermetropic. In hypermetropia there is a disproportion between the length of the eye and the curvature of the cornea, and it is not easy to say whether at birth the hypermetropia is an axial or curvative hypermetropia. The hypermetropia, which persists after adult life has been attained, is, however, almost always axial, and not only is the antero-posterior axis abnormally short, but the whole eye is often smaller than normal. In the higher degrees, indeed, there appears to be a kind of arrest of development, and this is often associated with a defective visual acuity. The acuity of vision in the lower degrees of hypermetropia is usually normal.

Hypermetropes, in whom the defect in refraction is not corrected, are necessarily obliged to exert more constantly, and to a greater extent, their power of accommodation than is the case with emmetropes. One consequence of this is that the ciliary muscle is usually very strongly developed in hypermetropes, and on this account perhaps the strain is less felt than it would be by an emmetrope suddenly rendered hypermetropic. Nevertheless the condition of refraction is frequently associated with a feeling of tiredness or pain, mostly referred to the supraciliary region, and coming on when the eyes have been used for a longer or shorter time for reading or other work near at hand. This is called *accommodative asthenopia.* The circumstances which influence the occurrence and severity of this symptom are —(1.) The degree of the hypermetropia; (2.) the age of the individual; and (3.) the state of the general health. The higher the degree of hypermetropia, so long as there is a possibility of accommodating at all for the distance at which the objects engaging attention are placed, the more likely is there to be asthenopia; and again, with the same degree, troubles are more frequent the older the individual, because the range of accommodation diminishes with age. Difficulties, too, which are overcome without much effort in a state of robust health, give rise to a much more conscious effort in conditions of physical and mental weakness or fatigue; consequently accommodative asthenopia is, *ceteris paribus*, more frequently complained of in the anæmic and delicate than in the robust and active.

When the range of accommodation is much restricted after middle age, or owing to paresis of accommodation, the difficulties of a hypermetrope are increased, as he then not only has a difficulty in seeing distinctly objects near at hand, but has also defective vision for a distance. This condition, in which the hypermetropia is not corrected by accommodation, is called *absolute* hypermetropia.

There is often noticeable in hypermetropes an apparent divergence of the eyes. This is due to the visual axis of the hypermetropic eye passing considerably to the inner side of the optic axis.

In a large percentage of cases of convergent strabismus there is found to be hypermetropia. The connection between hypermetropia and convergent squint is explained in the chapter on Strabismus. Many hypermetropes only occasionally use their power of accommodation to correct their distant vision, and thus do not constantly keep up the state of contraction of the ciliary muscle. They are in the habit of contenting themselves with an imperfect image of surrounding objects, unless anything should specially attract their attention.

Owing to the tendency, which exists in by far the greatest proportion of eyes, for the antero-posterior axis to become gradually elongated during the period of growth, an existing hypermetropia is often diminished in degree or converted into emmetropia, and even myopia, by the time an individual has ceased to grow.

The *treatment* of hypermetropia consists in the wearing of convex glasses. The strength required is that which corrects the manifest portion of the hypermetropia. The convex glass chosen is therefore the strongest with which the individual retains the full visual acuity he possesses. They should be worn constantly, for a distance and near, if asthenopia be complained of, as only by so doing is sufficient support given to the ciliary muscle. When there is no asthenopia, and the hypermetropia not of high degree relatively to the age of the individual, it is generally sufficient, if glasses be used at all, to make use of them for near work. They may then be ordered of a rather greater strength than that which corresponds to the manifest hypermetropia.

Patients with hypermetropia should be seen from time to time, at intervals say of a year after they have first begun to wear the prescribed glasses, in order to see whether there is any change in the amount of their manifest hypermetropia. In many cases there is none during the years of growing, at any rate, though as they get older the degree corrected constitutes often more and more of the total amount. In others there is soon found to be a further amount of the hypermetropia which is manifest, and which, when discovered, should be corrected. Again, in some children there is a pretty rapid diminution of the hypermetropia, so that after some time the glasses ordered have become too strong, and weaker ones should be substituted for them.

Hypermetropes have often small pupils, owing to the contraction of the pupil, which takes place along with that of the ciliary muscle.

By the constant use of convex glasses, which correct the manifest hypermetropia, the asthenopia often disappears after a short time. But we by no means find that this is always the case, even though we keep the accommodation paralysed for some weeks, and prescribe glasses which correct the whole, or nearly the whole, amount of the hypermetropia. The fact is, that the asthenopia, whether originally set up or not by the accommodative difficulties, is in many cases a nervous habit, which it is difficult to break,—more, therefore, a nervous and not merely an accommodative asthenopia. Marked permanent hypermetropia is probably always inherited, although the heredity is not always easily traced.

As has already been explained, the hypermetropia which occurs in old age is an *index hypermetropia,* produced by sclerosis in the crystalline lens.

The connection between hypermetropia and inflammation of the conjunctiva, cornea, and lids, has been much exaggerated. There can be no doubt that they are, originally, at any rate, completely independent affections, but it seems likely that they may be partly kept up by this state of refraction, as the use of correcting glasses appears not infrequently to have a favourable effect on the inflammatory condition.

There are no ophthalmoscopic appearances specially characteristic of hypermetropia. Sometimes there is an evident hyperæmia with some (usually slight) swelling of the optic papillæ. In a small proportion a white crescent is seen to the temporal side of the disc, due to the opening in the choroid, through which the optic nerve passes, being larger than the sclerotic opening, but this appearance is more frequently associated with the abnormally elongated eye.

MYOPIA.

Myopia is more frequent, and is met with in higher degrees in adult life than during the period of growth. It is hardly ever congenital, and usually first makes its appearance after the age of ten. It has a tendency to increase up till the time when the individual is fully grown, after which, in the vast majority of cases, it remains stationary. Progressive myopia, at a later period of life, appears to be more common in women than in men.

Myopia is mostly axial, and though not congenital, the tendency to the elongated shape of the eye is undoubtedly inherited. Except in the higher degrees (above 8 to 9 D) the visual acuity is in most cases normal, and the eye in every respect healthy. The visual acuity for near objects is generally greater in the uncorrected myopic eye than in the accommodated hypermetropic or emmetropic eye.

When uncorrected, the vision for distant objects is indistinct, and the more so the higher the degree of myopia. Uncorrected myopes, however, with the same degree of refractive error, do not by any means see equally badly at a distance, as much depends on the size of the pupil, and more perhaps on the habit, which some possess to a much greater degree than others, of making much out of a bad image. Thus we invariably find that those, otherwise intelligent and observant, who are not in the habit of wearing concave glasses, make more of the images, *i.e.*, arrive unconsciously at a more perfect cerebral elaboration of the images they receive of distant objects, than those who are constantly in the habit of receiving distinct images by the aid of correcting glasses.

The fraction representing the visual acuity is greater, too, the nearer the test object lies to the far point of the eye, and this is most noticeable in cases where the degree of the myopia is only moderate. Thus while V tested at 20 feet may be only $\frac{1}{10}$, it may be $\frac{1}{8}$ to $\frac{1}{6}$ at 10 feet, and $\frac{1}{6}$ to $\frac{1}{4}$ at 5 feet. *In this way the defective vision caused by myopia differs from amblyopia.*

The pupils in myopia are usually larger than normal, so that the defect of distant vision is in this way increased. The reason of this is, no doubt, the absence of the contraction of the ciliary muscle, which, from there seldom being any necessity for accommodation, is not brought into play so much as in the case of the other conditions of refraction. The ciliary muscle in myopic eyes is often found to be feebly developed, the circular fibres more especially being conspicuous by their absence.

Myopes have often a sleepy appearance, owing to the imperfect manner in which they see surrounding objects at any distance. For the same reason there is often a poking of the head forwards, a shyness of character, and a more marked fondness for reading and study than is evinced on the whole by those, the state of whose refraction enables them to see more distinctly surrounding objects or individuals. Hence in great measure it is that the number of myopes in the learned professions, and amongst those engaged in occupations necessitating near vision, is greater than is to be found amongst those who are otherwise occupied.

There is sometimes an apparent convergent squint in myopes, due to the circumstance that the visual axes pass slightly to the outer side of the optic axes.

Myopia is sometimes also the result of disease, and when this is the case, it as a rule rapidly assumes a high degree, and often eventually leads to blindness.

In some cases of myopia the absence of accommodative impulses leads to an insufficiency of the power of convergence, which is intimately associated with accommodation. Whilst the associated movements to either side are effected in such a manner as to show that there is no loss of power in the interni, the two interni muscles are unable to act together to the extent or with the ease that is required for comfortable work at a distance at which the eyes receive distinct images. This *insufficiency of convergence* is often wrongly called insufficiency of the interni. It gives rise sometimes to a feeling of tiredness when the eyes are used for reading,—what is called *muscular asthenopia*, of which it is one of the causes. The same impediment in the performance of convergent movements is apt, under circumstances of unequal acuity of vision in the two eyes, to give rise to divergent squint, a form of squint more frequently associated with myopia than with other conditions of refraction.

There is frequently an ophthalmoscopic appearance to be met with in the myopic eye, which, although not absolutely characteristic of this state of refraction, is more frequently connected with it than with such as are due to a less elongated form of the antero-posterior axis. The appearance referred to is that of a white patch, usually concentric in shape, immediately to the outer side of the entrance of the optic nerve. In some cases it is seen above or below instead of to the temporal side. This patch is due to an absence of some, or all, of the layers of the choroid in this situation, and a consequent reflection of more or less light by the white sclera. Its size bears no invariable relation to the degree of myopia. When large, *i.e.*, when its breadth is nearly equal to or exceeds the diameter of the papilla, and more especially when it extends all round the nerve, it is always associated with the higher degrees, and must, too, be looked upon as pathological, and suggestive of progressive myopia. The white *myopic crescent*, as it is called, is generally bordered with pigment where it meets with the edge of the normal choroid, and there is often at the same time an appearance of the central vessels of the nerve being pushed to the inner side, an appearance which is due to the edge of the aperture in the choroid coming closer up to the nerve than it does when there is no crescent on the other side.

In many cases of the higher degrees of myopia where the abnormal elongation is due to a staphylomatous protrusion of the back part of the eye, and consequently the myopia is the result of disease, the changes round the nerve are produced not only by displacement of the choroidal aperture, but by degenerative changes of an inflammatory nature as well. At the same time, other changes are met with in the region of the macula lutea. These, notwithstanding the existence of more or less metamorphopsia, or the appearance of distortion of objects seen, are at first not very evident, but as the myopia progresses, they usually make their appearance as a more or less indefinite irregularity in the retinal pigment layer at the macula, or it may be small whitish or yellowish-white spots in the same region. At later stages a ring-shaped pigmentation surrounding a more or less degenerated area, is often seen, and in extreme cases actual patches of considerable size may occupy the site of the macula or the retina immediately surrounding it.

The *treatment* of myopia consists, in the first place, and in all cases where it is merely a type of refraction, and not the manifestation of any disease, in wearing concave glasses. The glasses worn should as a rule be those which fully correct the myopia. Owing to the power of accommodation, stronger glasses than those required do not necessarily render the vision less distinct. The rule is therefore to give the *weakest* concave glasses which admit of the full visual acuity which the individual has.

When prescribed early in life, the correcting glasses may be worn both for distant and near vision without causing any discomfort, and with the advantage of rendering unnecessary the same degree of convergence of the visual axes which the uncorrected myope of any considerable degree is in the habit of exerting when reading. This is not always the case if glasses are first worn later in life, and it is sometimes necessary, even before the age at which presbyopia shows itself in emmetropes, to use weaker glasses for reading. This is probably owing to the unusual relation which is all at once introduced between the convergent and accommodative movements. Thus, whilst the individual has hitherto been converging up to the distance of his far point without exerting any accommodation, he has now all at once to associate with every degree of convergence a similar degree of accommodation.

The diminishing effect of the concave glasses, which is partly real and partly apparent, though disagreeable at first, generally very soon passes off, and in the lower degrees of myopia is indeed often not noticed at all. The same may be said of the distortion of larger objects to which glasses give rise; indeed, it is most remarkable that so little notice should usually be taken of this.

In progressive myopia due to disease great care should be taken to avoid any undue convergence of the axes, reading in a bad light, exposure to strong light, stooping, &c., anything in fact which might be likely to determine or keep up a congestion of the eyes. At the same time attention should be directed to the general health. Counter-irritation and local blood-letting are also recommended and may sometimes be of use.

The question as to the *etiology* and pathology of myopia is one of no little difficulty, and one on which many different views are entertained. The greater frequency of this state of refraction in some countries compared with others equally civilised, and the almost complete absence of it in uncivilised countries, has naturally given rise to the idea that it is associated with some of the conditions of civilised life which are more prevalent in some parts than others. No doubt certain circumstances to some extent influence the increase of myopia during the period of growth. Eyes at that time appear to have a tendency to adapt themselves to the conditions under which they are used. But great confusion has been introduced by a failure to discriminate between the cases of short sight due to disease, and those much more numerous cases which are the expression of a particular type, and are no more to be looked upon as pathological than is, for instance, the attainment of a greater than average stature.

The fate of many cases of the malignant forms being assumed as the possible fate of all cases of myopia, has given rise to all sorts of theories, supported either by statistics, anatomy, or experiment, to account for the progression which takes place during the period of growth, and to a corresponding number of more or less unpractical proposals as to how such progression is to be stopped. Attention has naturally been directed to schools, from which statistics, mainly from America, Germany, France, and Switzerland, have clearly shown that the percentage of cases, as well as the degree of short sight, increases regularly from the lowest to the highest forms, the increase being greater amongst the children of the better class schools than amongst charity school children. This has been taken as a proof of the adverse influence of school work. The main blame has been attached to various causes, and all the possible and impossible factors have been upheld as the most important. Some good has undoubtedly arisen from this agitation, as it has led to much-needed reforms in foreign schools in the way of better ventilation, better light, better desks, and more particularly greater attention being shown to physical training.

Not only have different conditions associated with the use of the eye at near distances been supposed to give rise to myopia, but many different explanations of the manner in which any particular condition brings about the elongation on which the myopia depends have been advanced. To take but one example: excessive convergence of the visual axes, which is looked upon by many as the main cause of the development and advance of myopia, is by some explained as acting by causing a dragging on the optic nerves; by others, by introducing either increased intraocular tension or a stasis in the circulation, resulting from the pressure of the external recti on the eye-balls. Some believe, too, that this may occur without the co-existence of any actual diseased condition of the coats of the eye at the region, which becomes gradually more and more distended; whilst others hold that this is impossible, except on the assumption, for which they find more or less histological evidence, of a low form of inflammatory tissue change. The myopic crescent has often, too, been taken to be the forerunner of a staphyloma posticum, whereas there is good reason to believe that in most cases it is of no pathological importance whatever.

The position of our knowledge with regard to myopia appears to me to be as follows:—We know that this state of refraction comparatively rarely begins much before puberty, that it increases regularly with age till the period of full growth has been arrived at. We know, further, that this increase takes place independently of schooling, and the numerous statistics which have yet been published, though strongly suggestive of a direct influence of school work on the progression, have by no means clearly established the existence or extent of such an influence. Even should it exist, there would be no reason why any pathological condition should be inferred in eyes which yielded to it, as it has been abundantly shown, and is the experience of any one who has paid attention to this question, that, notwithstanding the continuance of the same conditions as to reading, &c., in after life, the rule is for the myopia to remain stationary. The natural conclusion is, then, that if an influence is proved to exist at all, it is the expression merely of an adaptation of the eye to the work it is mostly called on to perform, occurring at a time of life when such adaptations are possible. One point of importance is, that although the educated classes present a far larger proportion of myopic individuals than the uneducated, still it is amongst the latter that the largest proportion of the higher and more complicated forms are met with. This fact is also an indirect evidence in favour of the ordinary myopia being in no sense a disease. The malignant form is relatively much rarer than the statistics of any ophthalmic clinic would lead one to suppose, owing to the circumstance that so many myopes of the ordinary type, having no retinal defect, readily find out how to correct their refraction themselves. We find, too, an undoubted hereditary predisposition to ordinary myopia, whereas little or none seems to exist in connection with the malignant form. This heredity is markedly visible in America, where, notwithstanding the subjection to the same conditions of school life, there is a great difference in the percentage of myopes found amongst the children of German, English, or Irish parentage. It appears to be a pretty definitely established rule that those countries which have longest been civilised have the largest proportion of myopes, and that, too, to a large extent independently of whether or not at the present time they are the most advanced in an educational point of view. Evidently, then, the adaptation that has taken place in the eyes to the requirements of civilisation in the way of early education has been slow, and probably influenced more by heredity than by individual application. We have no statistics to show in how far the condition of short sight has acted on the choice of a profession, and consequently those which demonstrate the preponderance of myopia in the learned professions are far from being conclusive, as they are generally taken to be, of the influence of study in causing or increasing it. In a society in which the possession of a certain amount of education is a requirement of the higher classes, the ordinary laws of natural selection must be at work, causing a preponderance of myopia in these classes. On the other hand, in savage communities, where so much depends on the possession of good distant vision, the same laws must act towards preventing the perpetuation of, or even actually exterminating, the myopic type. The higher degrees of hypermetropia appear also not to be met with in savage communities. The same law of the survival of the fittest would act against the perpetuation of this type.

A considerable difficulty, it must be confessed, is now and then experienced in determining whether, in any particular case, the myopia is to be looked upon as pathological or not, so that it is not always easy to draw a sharp line between the two forms. This is not to be wondered at, as the short sight at the time of examination may be the only symptom of what is actually a disease.

Whatever be the cause of the probable increase in the existing tendency to progression in myopic eyes during the period of growth, which application of the eyes to near objects produces, there can be little doubt that the proper treatment consists in wearing the correcting lenses. Any operative treatment, such as tenotomy of the external recti, paracentesis of the cornea, sclerotomy, &c., the indication for which rests entirely on theoretical considerations, is altogether unjustifiable in the present state of our knowledge.

ASTIGMATISM.

In the conditions of refraction hitherto discussed, we have seen that the rays proceeding from an external point met, after refraction through the transparent media of the eye, at some other point, or as nearly at a point as the existence of spherical and chromatic aberration admitted; that is to say, the refraction was symmetrical round the optic axis. When this is not the case the eye is said to be *astigmatic* (*α, στιγμα*), the name first given to this condition of refraction by Whewell.

Two forms of astigmatism occur. The want of symmetry may be of such a nature that the greatest refraction of the rays takes place in one plane, whilst the least refraction takes place in the plane at right angles to that plane, the refraction in intermediate planes being regularly intermediate in amount (though different in nature). This is what is called *regular astigmatism.* The cause is mostly a difference in the curvature of different meridians of the cornea, a difference which may be congenital or acquired. In highly astigmatic eyes the whole globe appears more or less evidently flattened in one direction, so that the deformity does not exist in the cornea alone. Regular astigmatism admits to a certain extent of optical correction.

When the meridians of greatest and least refraction are not at right angles to each other, there is what is called *irregular astigmatism.* This condition rarely admits of optical correction to any extent, and is caused by an irregularity in the curvature of cornea or lens.

The character of a regular astigmatism differs according to the position of the principal foci of rays refracted through the meridians of greatest and least curvature. If the focus of either coincides with the layer of percipient elements of the retina, the astigmatism is *simple.* Two forms of *simple astigmatism* occur: (1.) *Simple myopic astigmatism,* and (2.) *Simple hypermetropic astigmatism.* In the first, the focus of rays passing through the most highly refracting meridian of the cornea is situated in front of the retina, whilst that of the rays passing through the meridian at right angles coincides with the retina.—(Fig. 139, A.) In the second, the focus of rays passing through the feeblest refracting meridian is somewhere behind the retina, and that of the most highly refracting meridian on the retina.—(Fig. 139, B).

The astigmatism is *compound* if neither of these foci coincide with the retina. *Compound astigmatism* occurs in three forms.

(1.) *Compound myopic astigmatism,* when both the focus of rays through the highest and that through the lowest refracting meridians lie in front of the retina.—(Fig. 139, C).

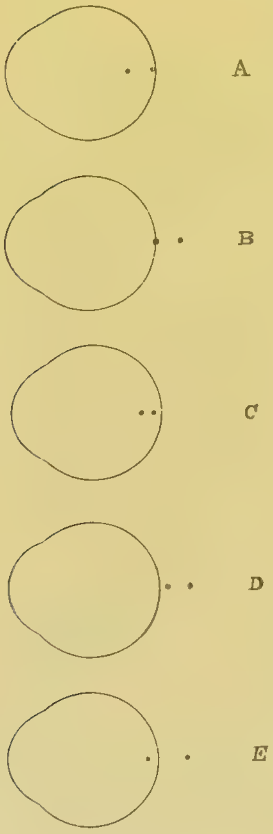

Fig. 139.

(2.) *Compound hypermetropic astigmatism,* when these foci are both behind the retina—(Fig. 139, D); and

(3.) *Mixed astigmatism,* when the one focus lies in front and the other behind the retina.—(Fig. 139, E).

Mixed astigmatism occurs much less frequently than the other forms. The compound forms are the most common. According to some, hypermetropic is more common than myopic astigmatism, whilst others have found the two about equally prevalent. In this respect there are evidently local differences just as in the proportion of myopia.

A certain degree of astigmatism exists probably in all eyes, but it is only when the difference in refraction in planes at right angles to each other is at least 0·50 D that an optical correction is called for. The correction is made by means of cylindrical lenses, or lenses which are portions of a cylindrical surface, and in which there is therefore one direction not curved, corresponding to the axis of the cylinder. This direction is called the *axis* of the cylindrical lens. The direction at right angles to this is that of greatest curvature, and the only one in which the curve is circular. Fig. 140 shows the plano-cylindrical glass, convex and concave.

For the correction of astigmatism the axis of the cylinder is placed parallel with that meridian, which, in the case of simple astigmatism, is emmetropic, and which, in the case of compound astigmatism, has been rendered emmetropic by a spherical lens.

The vision of astigmatic individuals is more or less distinct according to the degree of the astigmatism and the nature of the objects looked at. For ordinary print, for instance, and for most external objects, whose characteristic directions are vertical and horizontal, the direction of the chief meridians makes a difference. If the vision be tested for lines radiating in all directions, it will be found that in simple astigmatism lines parallel to the ametropic meridian can always be seen distinctly. The reason of this is obvious, as the distinct vision of a line mostly depends on the accurate focussing of rays which proceed from every point on it in planes which are at right angles to its direction. In compound hypermetropic and mixed astigmatism lines in some particular direction can also usually be seen distinctly, owing to one of the meridians being corrected for by accommodation. The lines which appear most indistinct are those at right angles to the clear ones. Sometimes the lines at right angles to the hypermetropic meridian appear most distinct owing to the emmetropic meridian having been rendered myopic and the hypermetropic meridian emmetropic by an accommodative change.

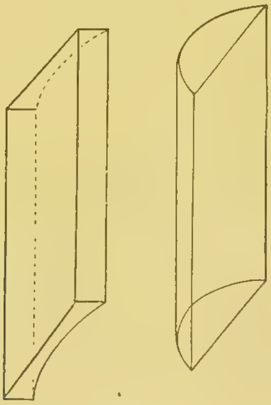

Fig. 140.

The eye is mostly accommodated for the focus of the rays passing through one of the principal meridians instead, *i.e.*, for one of the focal lines. There appears to be a preference for focussing the vertical focal line, and this is possibly, as Javal, who first called attention to it, suggested, owing to the possibility of at the same time improving the vision for horizontal lines by closing the lids.

The line most distinctly seen in compound hypermetropic astigmatism usually (though not always) corresponds in direction with the most ametropic meridian, and in mixed astigmatism with the myopic meridian. In compound myopic astigmatism no line is seen very distinctly, and the concave glass, which makes one line distinct, is the measure of the amount of myopia in the least myopic meridian, the direction of the line most distinctly seen being that of the more highly myopic meridian.

The manner in which those who are congenitally astigmatic see, is not very easy to understand. By holding a cylindrical lens in front of one's own eye, one may produce a distortion of images pretty similar to that which any one with the corresponding degree of astigmatism must receive on his retina. The fact, however, of this distortion having always existed makes it impossible for him to be conscious of it. But the curious thing is, that the cylinder correcting the astigmatism rarely produces any appearance of distortion, so that, both with and without correction, objects appear of much the same shape, though blacker or more sharply defined with the correcting glass. The appearance of radiating lines in a fan must be much the same as that produced by holding a cylinder in front of a non-astigmatic eye.

The *diagnosis* of astigmatism may be made both subjectively and objectively. As in the case of the diagnosis of myopia and hypermetropia, the former is the more satisfactory if the patient be at all intelligent, and the visual acuity on correction not much below the normal; but objective methods for the testing of astigmatism are less likely to lead to errors in the estimation of the amount of astigmatism present than in the other conditions of ametropia. Their uncertainty lies more in determining its position, *i.e.*, whether simple, compound, or mixed.

The subjective test for astigmatism is best made with cylindrical lenses and Snellen's test types, and, as far as the estimation of its degree goes, is easier and requires less knowledge of the theoretical and clinical aspects of refractive anomalies than the testing of myopia or hypermetropia. The reason of this is, that a cylindrical lens, if not of the proper strength, and not properly situated to correct the astigmatism, does not produce the desired improvement in vision, and is disagreeable owing to the distortion of objects to which it gives rise. But a cylindrical lens will often improve the ametropic vision to a considerable extent, although not required. On account of the visual disturbance being less in the case of astigmatism than in a similar ametropia of the same degree, one should always begin testing with spherical lenses. When it is found that spherical lenses produce no improvement at all, or only a certain degree of improvement as tested with Snellen's types, we may either proceed at once to try whether any improvement is to be got with cylindrical lenses added to the weakest concave spherical lens which gives the best vision, or the strongest convex one which does not make the vision any worse; or we may determine which of a number of radiating lines are seen most distinctly without any spherical glass, or with the addition of such a glass. In this way the refraction in one of the meridians is obtained, and if the form (Fig. 141) sometimes called "Snellen's rising sun" be used, an indication is afforded at the same time of the extent of visual acuity, which the addition of a suitable cylindrical lens may lead one to expect to find. After this cylinder is found, and the position in which its axis has to be placed—and this must be done by testing with which cylinder the vision is most acute—we must, by adding a weak convex lens, see whether some of the correction is not made by an accommodative change,

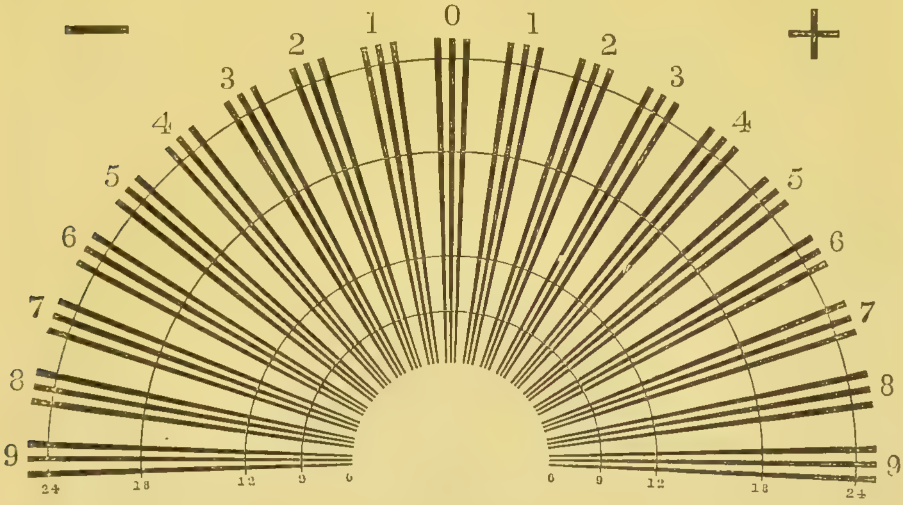

Fig. 141.—Snellen's test for astigmatism. Figure reduced nine diameters.

and eventually choose the combination of glasses in a case of compound astigmatism, in which the best vision is got with the weakest concave or strongest convex spherical lens.

A useful objective method—by which, however, the existence only, and not the amount, of the astigmatism can be diagnosed—consists in observing whether the image of the disc alters in shape as the lens used in making an indirect ophthalmoscopic examination is withdrawn from or approached to the eye. If it does so, there must be astigmatism. The reason of this will be readily understood from the explanation of the alteration of the size of the image obtained in this way given at page 508, and by a reference to Fig. 126. A difference in the state of refraction in different meridians of the eye causes the change in size to take place with different degrees of rapidity in different directions, or, in the case of mixed astigmatism, to increase in one direction and diminish in that at right angles, so that in either case an alteration in shape must take place. It is often convenient to make this qualitative test before proceeding to try the effect of cylinders; or if there be any doubt as to the change of shape on withdrawing the lens, and the papillæ appear oval when the lens is held close to the eye, we may proceed to ascertain its shape on direct ophthalmoscopic examination. If the oval is then at right angles to its former direction, there must be astigmatism. In this alternate examination by the indirect and direct we possess a delicate test for the existence of astigmatism.

The obvious reason for this rectangular difference is, that in the direct examination the most highly curved meridian of the eye examined produces the effect of a stronger magnifying glass, and therefore causes a drawing out of the image in that direction. By the indirect method of examination the stronger combination, which results from the more highly refracting meridian and the convex lens, gives rise to a correspondingly smaller inverted image. As a rule, the direction of the long diameter of the oval is vertical when seen in the direct, horizontal when in the indirect image. It follows from what has been said that, however oval a papilla may appear, it is not an indication of astigmatism, unless the shape differs by the direct and indirect methods of ophthalmoscopic examination, or on withdrawing the lens in the latter method of examination.

One of the simplest and best objective tests for astigmatism is made by retinoscopy. By noticing with which spherical lens a reversal of the shadow first takes place in one meridian, and then with which other glass the reversal takes place in the meridian at right angles to the first, which is done by rotating the ophthalmoscope mirror first round one axis, and then round that at right angles to it, we are able to determine very approximately the degree of astigmatism. It is evident that the difference in the strength of the lenses which first reverse the shadow in the two directions is the measure of the astigmatism, provided that there has been no change in the amount of accommodation during the examination. The position of the

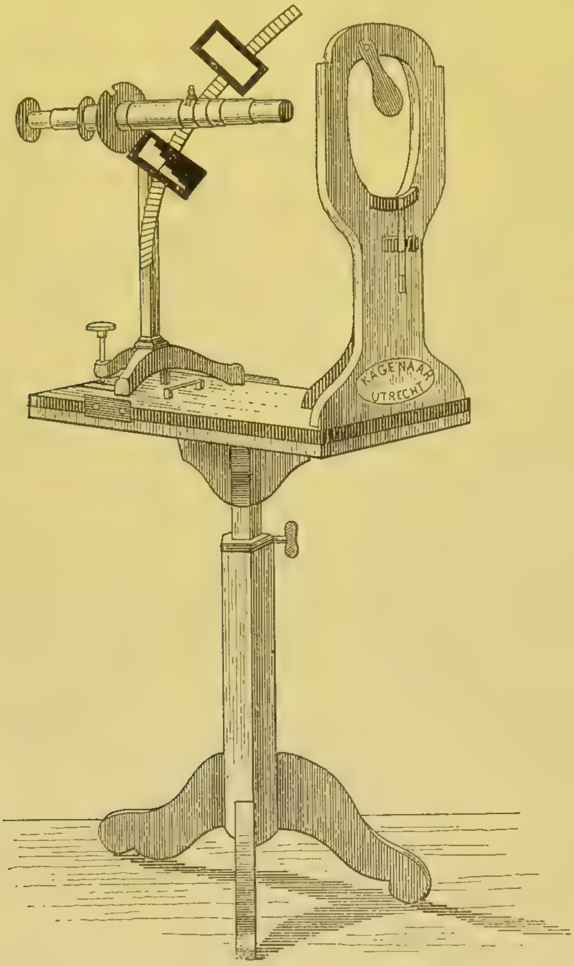

FIG. 142.—Javal's ophthalmometer.

astigmatism cannot, on the other hand, be got with certainty unless the accommodation be paralysed. When this is not done, it is necessary, after finding its degree, to ascertain by the ordinary subjective tests, when this is possible, the position, or at any rate the position which may most suitably be taken for purposes of optical correction.

It has already been said that regular astigmatism is mostly due to differences in curvature of the cornea in different directions. These differences in curvature can be ascertained by means of Javal's ophthalmometer, and the astigmatism calculated in this way. This instrument (Fig. 142) consists of a telescope placed horizontally and so arranged as readily to be directed on the cornea of the eye to be examined. Firmly fixed to the telescope is a graduated arc of 35 centimetres radius, the centre of which is in a line with the axis of the telescope, and therefore also with the visual axis of the patient when it coincides with that of the telescope. On either side of the midpoint of the arc are two movable white figures. These are the objects whose images on reflection from the cornea are seen magnified by the telescope. By means of two prisms joined at their apices suitably placed in the tube of the telescope the two corneal images are doubled. When the objects are placed at a certain distance apart, one of the images of the one object can then be made to appear to be close up to one of the images of the other object. This is seen in Fig. 143. The distance separating the two objects

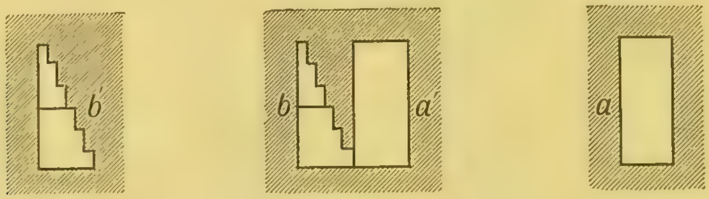

FIG. 143.

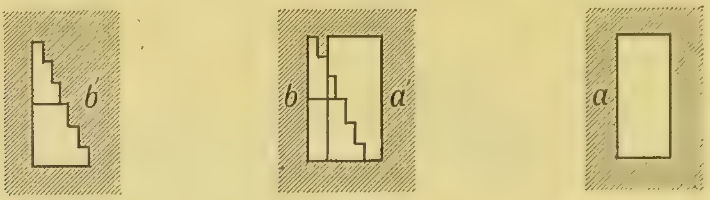

FIG. 144.

which produces this effect will depend upon the curvature of the cornea. Now, if in one position of the arc, say the horizontal, the white objects have been so placed that the one of their corneal images exactly touch, but do not overlap each other, and it is found that an overlapping takes place when the arc is rotated, so as to take up a position at right angles to its former position, evidence is thus afforded of a difference in the radius of curvature of the cornea in different meridians, *i.e.*, of corneal astigmatism. In Javal's instrument while the one object is rectangular, the other is so shaped, in steps, that for every step overlapping in the meridian of greatest curvature there corresponds one dioptre of astigmatism. This is shown in Fig. 144, which represents 4 dioptres of corneal astigmatism.

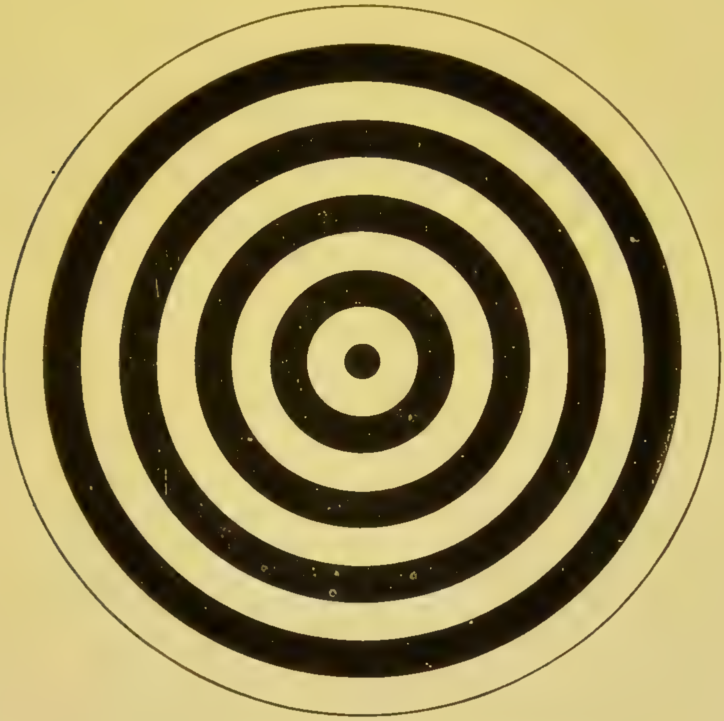

FIG. 145.—Placido's disc, ⅓ diameter.

Javal's optometer, though affording a simple and trustworthy means of calculating the amount of astigmatism, has the disadvantage of being expensive. Placido has devised a simple method, by which, however, only qualitative results are obtained, and the directions of greatest and least curvature shown at a glance. It consists of a piece of cardboard or sheet tin 8 to 10 inches in diameter, on which a number of alternately black and white concentric circular bands are painted from $\frac{1}{3}$ to $\frac{1}{2}$ an inch in breadth. A small hole is pierced in the centre of the disc, and the apparatus is used in the following way:—The individual to be tested is placed with his back to the window, and directed to look at the centre of the disc, which is held in the observer's hand and close up to his own eye, but at a convenient distance, not closer than the observer's near point from the eye of the individual examined. The observer sees through the hole in the disc the image of the concentric rings, which, if there be no astigmatism, appear circular; but if the curvature of the cornea be different in different directions, are elliptical in shape, the long axis, or the direction in which the image is largest, corresponding with that of least, the short with that of greatest, corneal curvature. If there be irregular corneal astigmatism, it is very beautifully seen by the use of such a disc. Care must of course be taken in using Placido's disc that its plane is at right angles both to the vertical and horizontal planes through the eye.

Many other subjective as well as objective tests for astigmatism are, or have been, in use, some of which need only to be referred to here, as the results are less satisfactory, or are only arrived at by the expenditure of more time than by the application of such as have been fully explained. Of the subjective tests more or less in use, may be mentioned the method by the use of stenopaic slits, which consists in determining by means of a narrow slit in front of the eye the position in which vision is most acute, and then taking as a measure of the degree of astigmatism the lens whose focal strength equals the difference between that which corrects in this position, and that which corrects for the position of the slit at right angles to the first. This method is principally defective on account of the tendency which there is to accommodate to different extents for the different positions of the slit. Then there is Tweedy's optometer, constructed on a principle, first used by Javal, which consists in first determining the distance at which with any particular convex lens a line parallel to one of the principal meridians of the eye is sharply defined, and afterwards the distance at which a line at right angles to the first is properly focussed. From the difference between these two distances the degree of astigmatism is calculated. This method is difficult, on account of the difficulty of being sure that the test lines are really properly focussed, but it is not altogether free from the difficulty of securing the same amount of accommodation throughout the examination. Stokes' lens, which consists of a concave and convex cylinder of equal strength, the axis of which may be placed in any position relative to each other, and thereby produce the effect of cylinders of varying strengths, has this defect, that although capable of correcting the astigmatism, it does so by producing the effect of a convex glass in one meridian and a concave in the other. Of objective methods, one frequently attempted is that of determining the refraction with the ophthalmoscope (erect image), first for vessels parallel with one meridian, and afterwards for vessels parallel with the meridian at right angles to the first. This method is not only tedious, and only possible after considerable practice, but is even more uncertain in its results than when used for the estimation of the degrees of ametropia.

It is usually the vertical meridian, or some meridian not far from the vertical on either side, which is most curved, and therefore the most myopic or least hypermetropic meridian; but the exceptions to this rule are numerous. Sometimes the ametropic meridians lie at right angles to each other in the two eyes, so that much of the visual defect in either eye is made up for when both eyes are used together. The amount of astigmatism is more frequently different in the two eyes. Though often the same, it is not always exactly so for near as for distant vision. Even when, as is most frequently the case, no appreciable difference in the astigmatism of the unaccommodated or accommodated eye exists, it is not always the cylinder which admits of the best distant vision that is found most suitable for near vision. Many, for instance, and especially if they are over thirty years of age, whose myopic astigmatism is corrected by a concave cylinder, prefer to use a convex one for reading, with the axis at right angles to the position in which the concave one has to be placed. The effect of this is, of course, to render the emmetropic, or more nearly emmetropic, meridian as myopic as the other, and not to neutralise the myopic meridian. The cause of this preference is not always, or even most frequently, connected with a habit of accommodating for a distance at which the rays passing through the meridian of greatest curvature are focussed, as might be supposed, as generally there is a preference given both for distant and near vision for the focussing of lines in the same direction. It is probably due to its introducing a more comfortable relation between accommodation and convergence. Most young individuals with myopic astigmatism seem to prefer, however, concave cylinders both for distant and near work.

The higher degrees of astigmatism (above 4·0 D) are not common. The highest I have met with as a congenital condition, has been 8·0 D, but still higher occur. In by far the greatest number of cases which call for correction the amount is from 1·0 to 4·0 D.

From what has been already said, it will readily be understood that the amount of improvement got by cylindrical correction varies very much in different cases, as this depends on the direction of the astigmatism, the intelligence of the individual, and the amount of possible visual acuity in each case.

To understand the nature of refraction in an astigmatic eye, we must consider what must be the course of the rays proceeding from a point after refraction at a surface, the curvature of which differs in different directions. In Fig. 146 let SS′ and $\sigma\sigma'$ be respectively the meridians of greatest and least curvature, SS′ being vertical and $\sigma\sigma'$ horizontal. Now, if we take the rays from an infinitely distant point which are refracted in these meridians, we have f'_1 and f'_2 the respective foci of such rays lying at a definite distance from the surface. The interval separating f'_1 and f'_2 is called the *focal interval.* At f'_1 a horizontal distance, $x\,y$, separates the extreme rays refracted through the meridian $\sigma\sigma'$, and at f'_2 a vertical distance, $x'\,y'$, separates the extreme rays refracted through the surface in the plane of SS after they have met and crossed at f'_1. $x\,y$ is called the *anterior focal line* and $x'\,y'$ the *posterior focal line* of the refracting surface. It can be shown that all rays from the distant point pass through these two focal lines, or more correctly through elongated figure of 8 areas which may sufficiently approximately be looked upon as lines. These lines are therefore the sections, at the distances of the foci of the principal meridians, of the circular bundle of rays which meets the surface parallel with its axis. At other distances this section has another form, and from being a horizontal line, becomes successively an ellipse with the long axis horizontal, a circle, an ellipse with the long axis vertical, and a vertical line. These forms of the section of refracted rays are indicated in the figure.

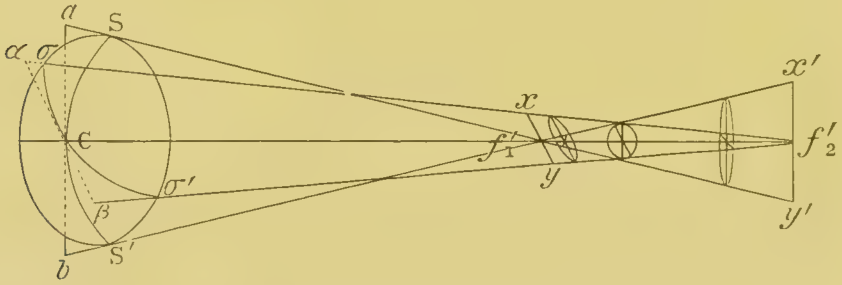

FIG. 146.

Without introducing any great error, we may take $a\,b = a\,\beta = z$,

and
$$\frac{z}{c\,f'_1} = \frac{x'\,y'}{f'_1 . f'_2}$$
$$\frac{z}{c\,f'_2} = \frac{x\,y}{f'_1 . f'_2}$$
$$\therefore \quad \frac{x\,y}{x'\,y'} = \frac{c\,f'}{c\,f'_2} .$$

That is, the anterior focal line is to the posterior focal line as the short focus is to the long. The anterior focal line is therefore always shorter than the posterior. The position of the circular section is therefore nearer the anterior than the posterior focus. If we call the diameter of the circle d, and its distance from f'_1, m_1, and from f'_2, m_2 we have
$$\frac{d}{m_1} = \frac{x'\,y'}{f'_1\,f'_2} \text{ and } \frac{d}{m_2} = \frac{x\,y}{f'_1 . f'_2}$$
$$\therefore \quad \frac{m_1}{m_2} = \frac{x\,y}{x'\,y'} = \frac{c\,f'_2}{c\,f'_2} .$$

From this we see that the greater the difference in the two focal distances the nearer relatively does the circle of diffusion lie to the anterior focus.

Only the rays refracted in the planes in which the centres of curvature of the two principal meridians lie meet at a point. It is for this reason that the meridians of greatest and least curvature are called the *principal meridians.* Those rays which meet the surface along any other meridian pass each other without cutting, for the reason that the normals to all other meridians do not lie in the same plane. On this account an astigmatic eye, whatever the position of the astigmatism, can only be accurately focussed for lines in two directions which are at right angles to each other.

One effect of cylindrical correction is that the magnification of the retinal image is unequal in the meridians for which the correction is made. That this is the case will be apparent by reference to the remarks on relative visual acuity at p. 531. A curvature myopia may be looked upon as giving rise to the same shortening of the anterior focal distance (compared with that of the typically built emmetropic eye) as is caused by an accommodative myopia of the same degree. The absolute visual acuity in the myopic meridian in the case of myopic astigmatism, corrected by a cylinder at the anterior focus of that meridian, will be less than the absolute acuity of the emmetropic meridian. This difference will depend upon the degree of astigmatism. It will be equal to the difference between the relative acuity of an emmetropic eye, accommodated for a distance corresponding to the far point of the myopic meridian of the astigmatic eye, and the absolute acuity of the same emmetropic eye.

In myopic astigmatism, the cylindrical correction, therefore, produces a diminution of the image in the myopic meridian, as compared with that of the meridian at right angles to it. For a similar reason the cylindrical correction in hypermetropic astigmatism leads to a magnification of the image in the hypermetropic meridian. It would thus appear that, as has recently been pointed out by Gullstrand, there must be a difference between the sizes of the retinal images, formed on cylindrical correction, of equally large objects by the meridians of greatest and least curvature, amounting to something like 1½ per cent. for every dioptre of astigmatism. In the case of simple astigmatism, this difference may, to a slight extent, be lessened by bringing the cylinder close to the eye. In compound astigmatism the degree to which this is possible with a sphero-cylindrical correction could hardly be appreciable.

It is no doubt greatly owing to this difference of magnification in different meridians that astigmatic individuals see objects differently on proper correction than they do without correction, *e.g.*, circles appear more or less distinctly elliptical, squares longer in one direction than another, &c.

Regular astigmatism is, as has been said, mainly corneal. The total amount of astigmatism found on a complete subjective examination rarely, however, coincides with that which the measurement of curvature of the principal meridians of the cornea by means of the ophthalmometer enables one to calculate as the degree of corneal astigmatism. There are several reasons for this. One which has been long known since Thomas Young demonstrated the existence of it in his own eye is a certain degree of *lenticular astigmatism* Donders found that the astigmatism due to the crystalline lens generally tended to counteract the corneal astigmatism. It does so when, as is the rule (see p. 533), the vertical meridian of the cornea has the greatest curvature. On the other hand, in corneal astigmatism contrary to the rule, that caused by the lens leads to an additional amount. For the same reason there may be astigmatism found subjectively where there is a complete absence of corneal astigmatism.

Lenticular astigmatism, whether it be manifest or masked by a contrary corneal astigmatism, is very rarely indeed at all high. It is due to a certain amount of obliquity of the lens whose axis does not coincide but makes an angle with the visual axis. This angle is greatest in the horizontal plane. Tscherning, who has recently measured it in a number of eyes, finds that it varies as a rule from 3° to 7°. The astigmatism produced by the greater obliquity is not much more than ¼ of a dioptre.

Another circumstance which leads to the corneal astigmatism, as estimated by the ophthalmometer, not corresponding to the subjective or what might be called the clinical degree of astigmatism, one, too, which is apparently of greater importance than the lenticular astigmatism, is the irregularity in the amount of curvature at different parts of the cornea. As a consequence of this, the degree of astigmatism necessarily varies with the size of the pupil, as was first pointed out by Tscherning. That determined by the ophthalmometer, on the other hand, is the same for the same eye whatever be the size of the pupil, as the reflections from which it is deduced always take place from the same points on the cornea; from small areas not very far removed from, and on either side of, the point at which the visual axis cuts the cornea. The astigmatism, as shown by the ophthalmometer, is in fact that which would be produced by the differences of curvature of a narrow zonular portion of the cornea (of 2 to 3 mm. in diameter) surrounding the visual axis. The measurements made by Helmholtz and others with the original ophthalmometer led to the conclusion that the surface of the cornea of a non-astigmatic eye was sufficiently approximately an ellipsoid of revolution, while that of an astigmatic eye was ellipsoidal. The calculations which led to this conclusion were based on only a few measurements at different parts of the cornea. As the visual axis was not, as a rule, found to coincide with the antero-posterior axes of the ellipsoid, the angle formed between these two axes received the name angle *α*. Quite recent measurements made by Sulzer with the more easily manipulated ophthalmometer of Javal have clearly shown that the form hitherto assumed for the cornea is not that which it really presents. With Javal's instrument it is easy to determine the curvature with great accuracy at many different points. The result of Sulzer's examination has been to show that in non-astigmatic eyes the central portion of the cornea is very approximately spheroidal. At about 15°, or on an average 2 mm. from the point at which the cornea is cut by the visual axis, a pretty abrupt increase in its radius of curvature begins to take place. From here onwards towards the periphery, the cornea is more nearly ellipsoidal, the eccentricity of the ellipsoid at any point increasing on passing towards the margin. There is further to be found a degree of want of symmetry in the cornea, as there does not appear to be an equal amount of diminution of curvature for equal distances along the two principal meridians, or even for equal distances along the two halves of the same meridian.

The extent to which these peculiarities of shape which characterise the great majority of corneæ influence the total degree of astigmatism, as compared with that found with the ophthalmometer, depends upon the size of the pupil, and upon the degree of its decentration. Sulzer found the centre of the pupil situated, as a rule, from 2° to 9°, or on the average 5° to the temporal side of the visual axis. The usual effect of the summation of the different degrees of astigmatism at different parts of the pupillary area of the cornea was found to be as follows:—(1.) That in eyes which do not show any astigmatism with the ophthalmometer, there is found subjectively a slight degree of astigmatism contrary to the rule. (2.) That in eyes showing with the ophthalmometer a low or moderate amount of ordinary astigmatism, the subjective astigmatism is found to be less, altogether absent or contrary to the rule according to the ophthalmometric amount, the degree of corneal asymmetry, the size of the pupil, and the extent of its decentration. (3.) That in eyes showing with the ophthalmometer an astigmatism contrary to the rule, there is found subjectively a higher amount of the same variety of astigmatism, owing to the influence of the temporal portions of the cornea for which the astigmatism is more pronounced; and (4.) That in eyes showing with the ophthalmometer a high degree of ordinary astigmatism, the subjective astigmatism is often found to be even higher.

Very soon after the ophthalmometer came to be generally used as a clinical instrument, the difference between the degree of total astigmatism as determined subjectively and that found with the ophthalmometer attracted attention. The explanation first given by Javal, and one which seems, notwithstanding its *a priori* improbability, to have received an astonishing amount of support, was that by an irregular or unequal contraction of the ciliary muscle, the lens could be rendered to a certain extent astigmatic, so as in some degree to overcome the corneal astigmatism. Before the question as to whether such partial contractions of the ciliary muscle really take place or not can be definitely settled, ophthalmometric measurements of the curvature of the lens at different points of its surface will have to be made. Tscherning has, however, pretty clearly shown, and his view has been confirmed by Sulzer, that the differences in question are mainly due to the shape of the cornea itself.

The importance of a consideration of this subject at all, apart from its scientific interest, lies in the practical clinical deductions which have been made on the assumption of the correctness of Javal's explanation. It has been held, for instance, that the supposed irregular contractions of the ciliary muscle, induced in the effort to contract a corneal astigmatism, are effected with an amount of strain which gives rise to headache and other nervous disturbances, to a tendency to increase of myopia, and even to corneal inflammations.

Headaches due to astigmatism.—While there can be little doubt that in some way or other astigmatism may give rise to headaches, it is equally certain that its influence in this respect has been greatly exaggerated. In my experience, cases of astigmatism contrary to the rule are more likely than the more common variety to cause headache. The higher the degree of astigmatism, and the more defective the vision either produced by or accompanying the refractive error, the more likely is headache to be complained of. In healthy individuals a slight degree of astigmatism up to 1·5 D is not at all likely to cause persistent headache. In delicate, overworked, neurotic people, on the other hand, it certainly seems as if the inconvenience caused by even a comparatively trifling astigmatism may tend to keep up the headaches of which they are apt to complain. In such cases it is right that the proper correction should be worn, even although one must be prepared often to find little benefit obtained by it.

IRREGULAR ASTIGMATISM.—A want of symmetry in the curvature of the lenticular or corneal surfaces gives rise to irregular astigmatism. This condition may be either congenital or acquired. The congenital is mostly dependent upon abnormal conditions of the crystalline lens; the acquired form, on the other hand, is mainly the result of alterations in the corneal curvature caused by cicatricial contractions following inflammations of that structure. Irregular astigmatism from partial dislocation of the lens is occasionally met with, and in such cases the refractive anomaly is partly due to the unsymmetrical curvature, and partly, as I have seen in some cases, to a wrinkling of the lens capsule.

The lenticular form, whether congenital or acquired, is often more pronounced for near vision, the defects of the lens being more marked when it has undergone an accommodative change. The sclerosis, which takes place, too, in the lens as age advances, tends to increase the existing defects on which the irregular astigmatism depends. This abnormality, besides producing more or less defective visual acuity, is occasionally associated with the consciousness of distorted vision (metamorphopsia) as well as with polyopia.

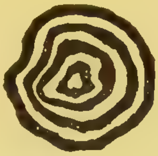

Fig. 147.—Distortion of Placido's rings on reflection from a cornea with irregular astigmatism.

Irregular astigmatism does not admit of optical correction, though many eyes which exhibit this defect are greatly improved by spherical or cylindrical lenses, owing to the co-existence of the more regular anomalies of refraction. Sometimes a stenopaic aperture opposite some particular portion of the cornea effects an improvement. According to Raehlmann, the weaker numbers of the hyperbolic lenses introduced by him for the correction of conical cornea are capable of improving the vision in some cases. My own experience, so far, with these lenses in irregular astigmatism has not been encouraging.

The diagnosis is readily made by the ophthalmoscope by the indirect method of examination. On withdrawing or approaching the auxiliary lens to the eye, the papilla is observed to assume various irregular shapes, giving rise to an appearance quite characteristic of this condition. If the irregular astigmatism be corneal, it is beautifully seen by the use of Placido's disc, with which instrument it is easy at the same time to discover the portion of the cornea, if any such portion exists, in which the curvature is least abnormal.

CONVERGENCE.

When an object situated at 6 metres distance is fixed by the foveæ of both eyes at the same time, the convergence of the visual axes on it is so slight (not $\frac{2}{3}$ of a degree) that they may be considered parallel. When nearer and nearer objects are fixed, a greater and greater convergence of the visual axes takes place. For an object at a distance of 1 metre from either eye, the angle at which the axes meet is almost $3\frac{1}{2}°$, for distances of $\frac{1}{2}$ and $\frac{1}{3}$ metre it is respectively 7° and $10\frac{1}{2}°$. Yet up to this point the convergence which has taken place is often not very apparent, owing to the very considerable angular separation frequently existing between the corneal axes and the lines of vision.

Under ordinary conditions it is evident that the requirements of distinct vision necessitate a convergence of the visual axes on the point accommodated for. In every case, therefore, in which binocular fixation takes place, there will be for every particular amount of accommodative action brought into play a definite degree of convergence with which this is associated. As we are now in the habit of expressing the refractive change which takes place on accommodation by the equivalent lens of the metrical system, it seems advisable to follow Nagel in applying a similar system of notation to the degree of convergence with which such a change is associated. According to Nagel's notation, when the eyes converge to a point 1 metre distant from either, the amount of convergence towards the middle line is taken to be 1 *metre-angle.* If the degree of convergence be such as to bring about fixation of a point $\frac{1}{2}$, $\frac{1}{3}$, $\frac{1}{4}$, or $\frac{1}{10}$ of a metre distant from either eye, the convergence required amounts respectively to 2, 3, 4, and 10 *metre-angles;* whilst a degree of convergence less than toward a point 1 metre distant is expressed as a corresponding fraction of 1 metre-angle—convergence to a point at 2 metres being $\frac{1}{2}$ a metre-angle, and so on. We have seen that, although individual differences exist in this respect, there is no hard and fast connection between the associated movements of accommodation and convergence; so that a certain amount of accommodation may be maintained with different degrees of convergence. Just in the same way it is found that a certain degree of convergence may be maintained with different amounts of accommodation. In Fig. 148 the N curve gives the maximum, the F curve the minimum convergence for every degree of accommodation in the case of a girl of thirteen.

Obviously very different relations exist between accommodation and convergence in the case of ametropia from those which may be looked upon as normal, and which exist when both eyes are emmetropic. A hypermetrope is obliged, if he retains binocular vision, to exercise more or less accommodation, according to the degree of his defect, before he is called upon to converge at all; whereas, on the other hand, a myope is so situated that convergence must take place before any accommodative change is called for. The theoretical and practical questions, raised by a consideration of the manner in which these associated movements are connected in different cases, are referred to in Chapter XVII.

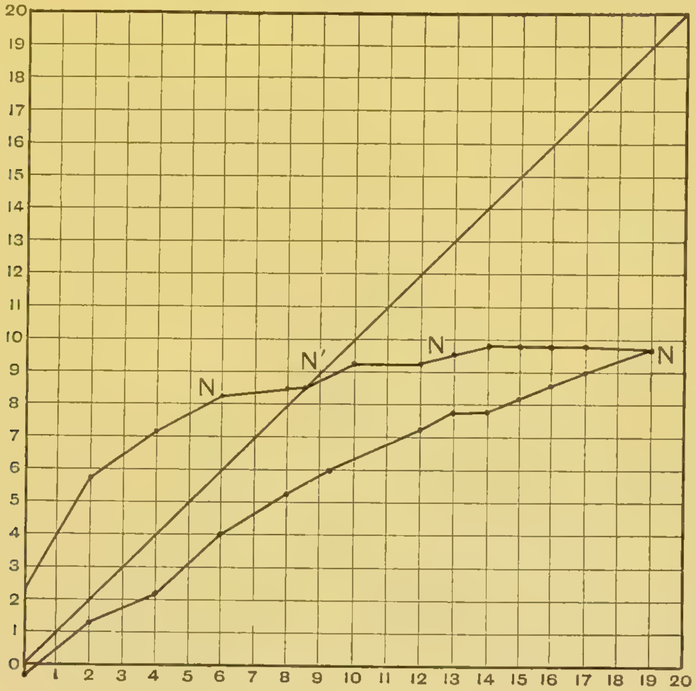

Fig. 148.—(After Nagel.)

Sometimes prisms are used in the form of spectacles, alone, or combined with spherical lenses, in order to ease the movements of convergence or divergence, or, where these movements are enfeebled, to stimulate them. Their use is limited, and they are rarely of practical value, on account of their weight and the chromatic aberration and astigmatism which they produce when at all strong.

The action of transparent prisms of greater density than the surrounding medium, and therefore of prisms of glass surrounded by air, is to refract the rays of light which pass through them in such a manner that their direction after refraction is more towards the base of the prism than that in which they are incident. Prisms with the bases outwards before one or both eyes therefore cause objects to appear nearer and smaller, owing to a greater degree of convergence being necessary to bring the rays from them on to both maculæ, than when the prisms are absent. Prisms with their bases inwards, on the other hand, cause objects to appear more distant and larger than they are, owing

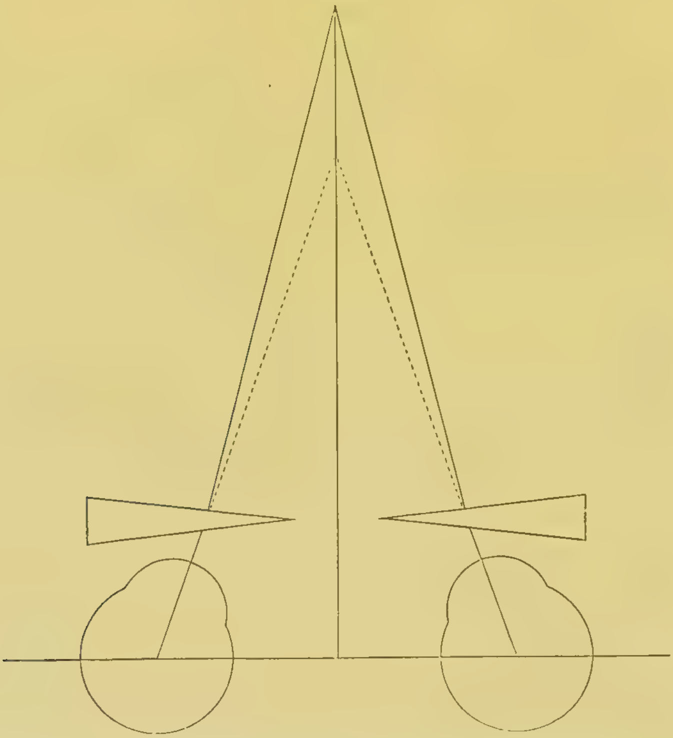

Fig. 149.—Position of prisms causing adduction and diminishing the necessity for abduction.

to the convergence required for their binocular fixation being lessened. Prisms in the former position therefore lessen the strain on divergent movements, and at the same time tend to induce greater convergent movements. Exactly the opposite is the case when they are placed in the latter position. This will be readily understood by a reference to Figs. 149 and 150, the continuous line being the direction of the axial rays from the object, the dotted ones the direction in which they appear to come.

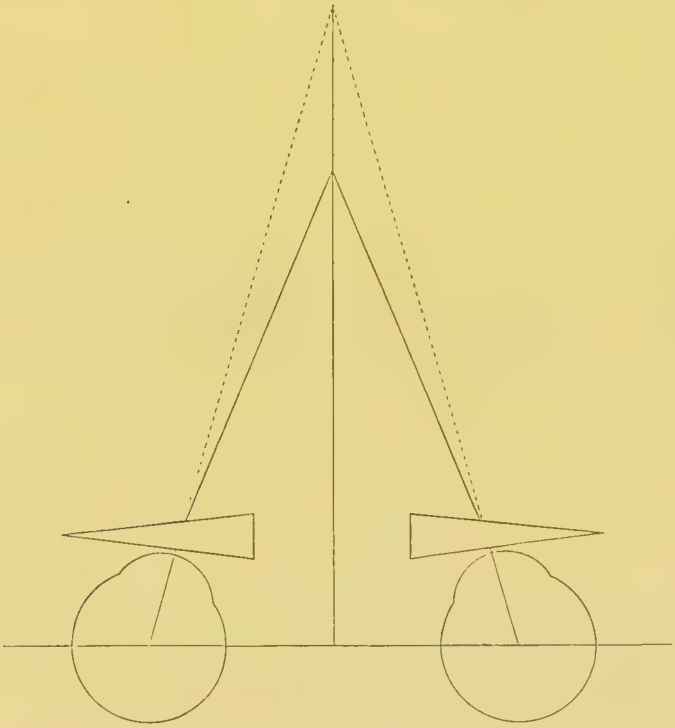

FIG. 150.—Position of prisms causing abduction and diminishing the necessity for adduction.

Prisms of 4° in front of each eye are about as strong as can conveniently be worn in most cases. Under special conditions considerably stronger ones are tolerated. The angular deviation produced by ordinary glass prisms is equal to half the angle of the prism; therefore, with 4° prisms in front of each eye, the deviation amounts to 4° in all.

Taking the case where both the angle formed by the two surfaces of the prism, *i.e.* the angle of the prism, and the angle of incidence, are small, which is the case that occurs in practice, we may determine the deviation in the following way :—

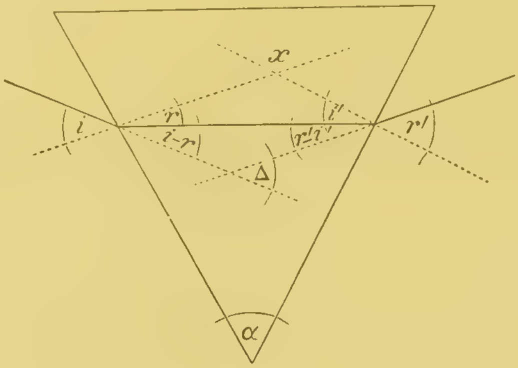

FIG. 151.

In Fig. 151 $\Delta =$ the deviation, i and r the angles of incidence and refraction at the first, and i' and r' those at the second surface ; $a =$ the angle of the prism, and $x =$ the angle between the normals to the two surfaces. From the figure we see that

$$\begin{aligned}\Delta &= i - r + r' - i' \\ &= i + r' - (r + i') \\ &= i + r' - a \quad . \quad . \quad . \quad \text{(A)}.\end{aligned}$$

And
$$\sin i = \mu \sin r$$
$$\sin r' = \mu \sin i'.$$

As in the special case considered all these angles are small, we may write for their sines the angles themselves ; we have

$$i = \mu\, r$$
$$\text{and} \quad r' = \mu\, i'.$$

Putting these values in (A), we have

$$\begin{aligned}\Delta &= \mu\,(r + i') - a \\ &= \ \ (\mu - 1)\, a.\end{aligned}$$

When $\mu = 1{\cdot}5$, as in the case of ordinary glass,

$$\Delta = \tfrac{1}{2}\, a,$$

i.e., the angle of deviation is equal to half the angle of the prism.

By decentering spherical lenses—that is, by so placing the lenses in front of the eye that their centres, instead of coinciding with the axes of vision, are more or less displaced outwards or inwards—a prismatic effect may be combined with that which is required for the correction of errors of refraction. The effect thus produced varies directly with the strength of the glass and the amount of displacement. In the case of convex glasses, displacement inwards towards the nose, and in the case of concave glasses displacement outwards, produces the effect of abducting prisms, *i.e.*, prisms with their bases inwards; while displacement of convex glasses outwards and concave inwards is equivalent to adducting prisms.

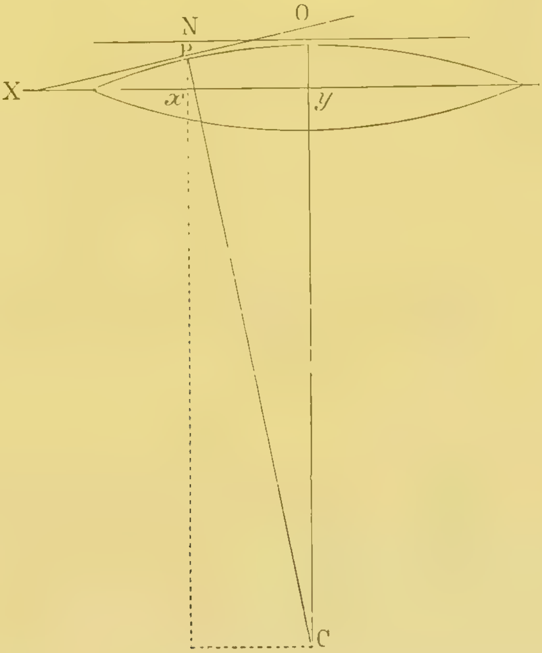

FIG. 152.

The increase in the prismatic equivalent of a spherical lens, as rays are refracted further and further from its axis, is seen by drawing tangents to different points on the surfaces equidistant from their common axis. When the two surfaces are of equal curvature, these tangents meet on the prolongation of their common chord, and the angle formed between them is the angle of the prism corresponding to the particular distance from the axis (Fig. 152). To calculate the deviation produced by any degree of decentering, we observe that $O C P = O X Y = \frac{1}{2}$ angle of prism (when the two surfaces are of equal curvature).

$\therefore O C P = \Delta$, and $x\,y$, or the amount of decentering may, without introducing an appreciable error, be put $= O N = R \tan O C P =$

r tan Δ. Putting d for xy, and f the focal distance of the lens for R, we have

$$d = f \tan \Delta.$$

Putting then m (Fig. 153) for half the interocular distance, and c' for the distance, along the middle line, from which rays from the point P appear to diverge, we have—

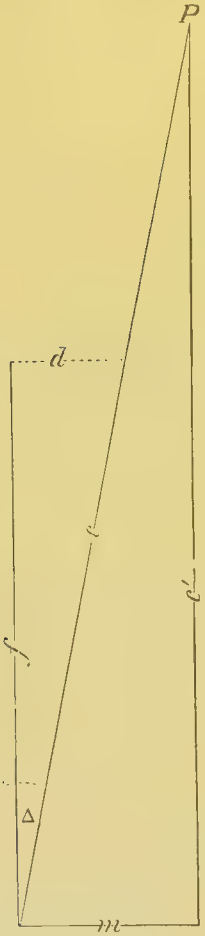

Fig. 153.

$$\frac{d}{f} = \tan \Delta = \frac{m}{c'}$$

and $c' = c \cos \Delta$, where c is the actual distance of the point P from the eye

$$\therefore \quad \frac{1}{c} = \left(\frac{1}{f} \cdot \frac{d}{m} \right) \cos \Delta.$$

For $\frac{1}{c}$ put M_C, and for $\frac{1}{f}$ put L_A, to denote respectively the number of metre angles of convergence, and the lens, whose focal distance is f, and the formula becomes

$$M_C = \frac{L_A \cdot d \cos \Delta}{m}.$$

When $d \cos \Delta = m$, $M_C = L_A$, the angle of convergence has the same metrical value as the lens, or the rays which come from the distance of the foci of the lenses in front of the eyes have a deviation as if they come from a great distance, and therefore no convergence of the eye is necessary for the binocular fixation of an object at that distance. Accommodation and convergence are aided to the same extent. Practically, Δ being small, cos Δ is so nearly $= 1$, that we may consider $M_C = L_A$ when $d = m$—*i e.*, a decentering of each convex lens to the extent of one-half the interocular distance gives the same assistance to convergence as it does to accommodation. This effect is produced by what are called orthoscopic lenses, or lenses which are portions of a large lens, at distances to either side of its axis equal to half the interocular distance, as represented in Fig. 154.

As an example of the above formula for decentering, suppose that with a 5 D lens it is desired to ease convergence to the extent of 1 metre angle, we should have, putting $\cos \Delta = 1$, and taking 60 mm. as the interocular distance—

$$1 = \frac{5\,d}{\cdot 03}$$

$$\text{or } d = \cdot 006.$$

That is, each lens would have to be displaced inwards to the extent of 6 mm., so that instead of the distance between their centres being 60 mm., it would be only 48 mm. A similar effect would be got by combining prisms of 4° with the spherical lenses.

A number of inconveniences are experienced by the wearer of spectacles, some of which depend on the altered physical, others on the altered physiological conditions which are brought about. Some of the apparent distortions complained of cannot be referred to any physical cause, and are in all probability, as has been pointed out by Nagel, due to a want of coincidence between the point at which the lines of external projection from the different parts of a retinal image cut each other, and the altered position of the second nodal point in the combined system of eye and spectacle. The altered relation between accommodation and convergent movements, already referred to, often introduces a difficulty when correcting glasses are first used late in adult life. Most of the difficulties experienced on first wearing glasses disappear, however, if the glasses have been properly chosen and are suitably placed in front of the eyes.

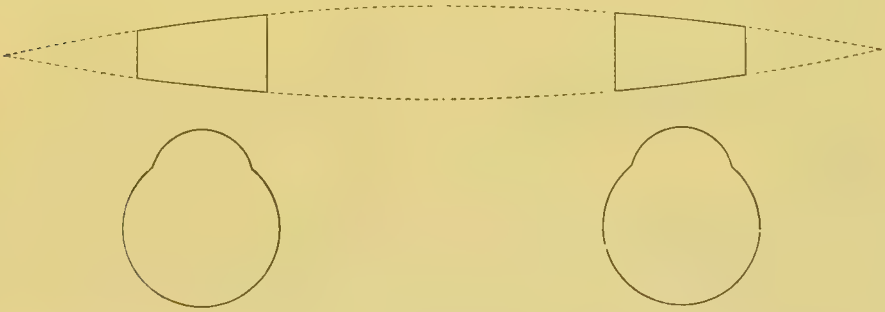

FIG. 154.

Owing to the prismatic action of the glasses, the field of vision taken in by a concave glass is larger, and at the same time more compressed; that taken in by a convex lens, on the other hand, is smaller and more dispersed than that seen through a simple aperture of the same size as the glasses. The doubling which this produces in the case of the concave glass of objects, rays from which enter the pupil both past and through the glass, and the corresponding breach in the continuity of the field of vision in the case of convex lenses, is, unless the glasses be small and strong, only noticeable as a rule if attention be drawn to it.

From what has been said of the decentering of lenses, it is evident that it is a matter usually of some importance to have the lenses properly centered. This cannot of course be done at the same time for all positions of the eye, but it is seldom that any appreciable interference with vision results from the oblique direction in which the visual axes cut the lens on looking to either side, although more or less astigmatism is produced in this way.

When the same glasses are worn both for a distance and near, they should be centered for distant vision. Reading glasses should be centered for the distance at which they are used, and should be at the same time slightly tilted in their frames, so that instead of being parallel with the face, they come rather nearer it below than above.

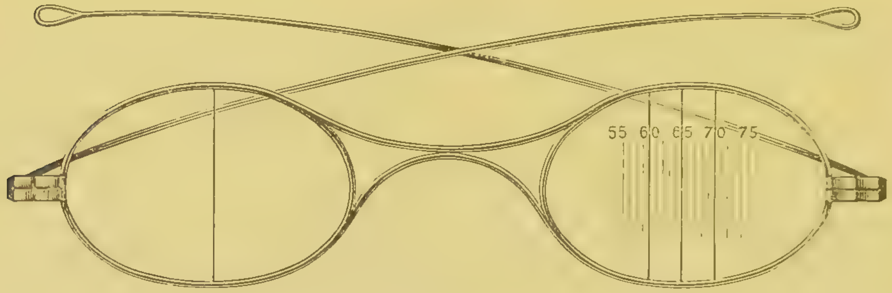

FIG. 155.—(Slightly reduced in size).

A simple method of measuring the interpupillary distance is by means of Snellen's spectacle frame (Fig. 155), which consists of a pair of plane glasses in an ordinary frame, with a vertical line scratched in the centre of one glass, and a number of lines 1 millimetre apart scratched on the other. By putting these on the patient's nose, and causing him to look at a distance, and in such a direction that with one's own left eye the line on the glass opposite his right passes through the centre of his pupil, and then observing which line on the other glass is seen by the right eye (on closing the left) to be opposite the centre of his other pupil, it is easy to measure quickly the interpupillary distance with sufficient accuracy for all practical purposes.

ANOMALIES OF ACCOMMODATION.

Reference has already been made to what may be called the passive defects of accommodation, viz., those which depend upon the absence of a suitable alteration in the curvature of the crystalline lens in response to a definite contractile change in the ciliary muscle. The chief defect of this nature—presbyopia—is physiological, inasmuch as it is a necessary consequence of the manner in which the growth of the lens takes place under normal conditions. In the various dislocations and sub-luxations of the lens, we have, on the other hand, the chief causes of pathological passive defects of accommodation.

More active accommodative defects arise from an interference with the function of the ciliary muscle. That muscle may be incapable of contracting at all, or its contractile power may be weakened.

Paralysis of Accommodation.—Complete paralysis is almost invariably the result of some lesion of the third nerve. Paresis, on the other hand, may be either a muscular or an innervation weakness.

Associated with complete paralysis we usually find, at all events, paralytic dilatation of the pupil; not infrequently also paralysis of the oculo-motor muscles supplied by the third nerve. These different paralyses are not so often found co-existing with partial paralysis of accommodation. Sometimes, however, there is at the same time a degree of paresis, or even complete paralysis of convergent movement, a movement which is usually associated with active changes of accommodation.

Roughly speaking, the affection which leads to paralysis of accommodation may be either central or peripheral. For instance, the paralysis may be one of the symptoms of all lesions of the brain—injuries, inflammations, tumours—which involve either the trunk or nucleus of the third nerve. Similar affections of the orbit, too, may, by involving some or all of the branches of the third, cause interference with the power of the ciliary muscle. A peripheral neuritis on the one hand, and disease limited to that portion of the third nerve nucleus in which is the accommodation centre on the other, may be the cause of paresis of accommodation alone.

Two of the most interesting clinical forms of isolated accommodation paralysis are the cases following concussion of the brain and diphtheria. I have seen several cases of more or less pronounced paresis of accommodation after railway accidents. The existence of such a paresis may indeed prove to be the most important evidence of shock sustained. In the case of a woman who was for some months a patient in the Royal Infirmary, almost complete paralysis of accommodation was apparently the only permanent injury resulting from a fall from an express train travelling at the rate of forty-five miles an hour.

Paralysis of accommodation following diphtheria is of pretty common occurrence. It is probably always bilateral, and rarely complete. The paralysis first makes its appearance some weeks after recovery from the diphtheria, at a time, too, when the patient has to a great extent recovered, and seems in other respects in good health. Occasionally it may be associated with other pareses, such as mydriasis, paresis of convergence, paresis of the sixth. Sometimes a concomitant convergent strabismus may exist along with the paresis of accommodation. Of other paralyses, that of the soft palate is much the most frequent.

The most notable feature in connection with the etiology of diphtheritic paresis of accommodation is that no evident relation exists between it and the severity of the preceding throat affection. Often, indeed, that has been so slight as to have been overlooked. It has even been asserted by some writers that the diphtheritic poison may lead to paralysis without having given rise to any local inflammation.

The *prognosis* in diphtheritic paresis of accommodation is more favourable than in paresis of accommodation from almost any other cause. It does not as a rule last longer than from four to six weeks.

The *treatment* should consist in giving tonics, especially iron and quinine. Myotics (eserine or pilocarpine), which are usually recommended, are of very doubtful value; possibly, indeed, they may do harm sometimes. I have always prescribed the temporary use of convex glasses of sufficient strength to enable the patient to read in moderation without any strain of accommodation.

Various views have been held as to the cause of diphtheritic paralysis. Probably the pathology of the rarer and less symmetrical paralyses may be different from the more common paralysis of the palate and ciliary muscles. There can be little doubt that post-diphtheritic accommodation paresis at all events is due to some toxic action on the brain. The bilateral character of the pareses, its transitory nature, the occasional complication with paresis of other associated movements are all suggestive of such a cause. Certain products of decomposition, particularly of fish, also cause paralysis of accommodation when absorbed from the alimentary canal. The only apparent difference between the paralysis arising in this way and the post-diphtheritic variety is that the former is more transitory. This probably is simply because the poison is more rapidly eliminated from the body. How the poison producing paralysis after diphtheria is formed or how it is eventually eliminated is not known.

Of the toxic effects coming under the notice of the ophthalmic surgeon that which perhaps most closely resembles diphtheritic paralysis of accommodation is the central amblyopia produced by tobacco (see p. 440). Both with respect to their bilateral character and their slow disappearance, there is a considerable resemblance between these two affections. In both cases it is unknown how the poison acts, whether directly on the centres or indirectly through some localised vasomotor change.

Weakening of accommodation owing to exhaustion of the ciliary muscle is a very frequent result of any severe debilitating illness. Many months may then elapse before the muscle regains its full power, or, at all events, is capable of prolonged efforts of contraction. The same symptom characterises most forms of neurasthenia. I have, for instance, seen many examples of the minor degrees of this form of paresis following influenza. The ciliary muscle may be overstrained, too, when prolonged efforts of accommodation are made under adverse conditions, such as the presence of corneal opacities, astigmatism, &c. The range of accommodation may thus be temporarily diminished.

Diminution of the range of accommodation may also be brought about reflexly, and in this way may arise from facial neuralgia, toothache, &c.

Whether partial or complete the greatest inconvenience from paralysis of accommodation is felt by a hypermetrope and the least by a myope. In hypermetropia the absence of accommodation causes an indistinctness of distant as well as of near vision. In emmetropia only near vision is interfered with, while in myopia of four or more dioptres the disturbance is so slight that it is apt to remain unnoticed.

It is not difficult to diagnose the presence of complete paralysis of accommodation even in the case of myopia, especially if the myopia admits of good correction. But if the myopia be considerable it may be difficult enough to feel sure of the existence of a paresis. In addition to innervation and muscular weaknesses, an interference with accommodation may result from inflammatory or atrophic conditions of the ciliary muscle. The most frequent example of this form of paresis is met with in glaucoma, of which affection it is not unfrequently an early symptom.

Spasm of Accommodation.—True, persistent spasm of accommodation is either of very rare occurrence or has no real existence at all. I have certainly never met with a case. I have not unfrequently, however, seen cases of so-called spasm of accommodation in which an attempt at seeing distinctly is apt to produce an excessive and unsuitable degree of accommodation. Such individuals if emmetropic are found only to see distinctly at a distance with concave glasses, and may thus be mistaken for myopes. If examined with the ophthalmoscope the true state of refraction is at once evident. This shows that the spasm is only induced when attention is directed to external objects. A similar contraction of the ciliary muscle takes place, in fact, to that which in a young hypermetrope leads to some of the hypermetropia being latent. The difference between the estimation of the refraction, got respectively by a subjective examination and direct ophthalmoscopic examination, affords the only means of diagnosing the condition with certainty, unless recourse be had to paralysing the ciliary muscle by the use of atropine. Not unfrequently the temporary spasm may be suspected before an ophthalmoscopic examination is made, owing to the manner in which the patient reads the distant test types. At the moment, if asked to read the smaller letters, he is quite unable to do so, but if he begins the next moment with the larger letters and finally passes through the series, he may momentarily exhibit a far greater visual acuity than he has, as a rule, without concave glasses. Those most liable to suffer in this way are young women whose occupation necessitates constant accommodation for near objects.

CHAPTER XVI.

THEORY OF THE OPHTHALMOSCOPE.

Owing to the shape of the eye and the relatively small opening formed by the pupil, much of that portion of the light passing into the eye, which is reflected from its inner surface, passes out in the same direction as it entered. On looking at anyone's eye the pupil appears black, because the observer's head intercepts the rays which would pass directly into the eye in the direction of the lines joining the head of the observer and the pupil of the eye observed. We cannot, consequently, under ordinary circumstances notice any illumination of the fundus, and therefore do not see any image of it. Only if the pupil looked at be very large, and the eye ametropic to some extent, is the red reflection as a rule apparent.

By means of the ophthalmoscope, which was invented by Helmholtz in 1851, we bring our eye in a line with the entering, and thus are able to catch the emerging rays. In the instrument as first invented the reflection was made at an angle of about 56°, from a number of pieces of plane glass enclosed at the sides in a darkened case, and open behind where the observer placed his eye, and caught as many of the returning rays as were not reflected again from the series of other surfaces. An improvement on this arrangement was shortly afterwards made by Ruete, by using a mirror with a hole in it, and a convex lens to obtain an aërial image. The mirrors used in the different forms of ophthalmoscope have been plane, concave, and convex. Reflectors which have consisted of combinations of differently-shaped lenses and mirrors have also been used. The plane and concave mirrors are now the only ones in general use.

The *intensity of illumination of the fundus* depends upon the luminosity of the source of light and the completeness with which the mirror used reflects the light; also upon the size of the pupil and the extent to which it is occupied by rays reflected into it from the mirror. The *apparent brightness* of any part, on the other hand, depends not only upon the actual intensity of its illumination, but also upon the extent to which the observer's pupil is filled by the rays passing into it from that part.

In considering the illuminating capabilities of different forms of ophthalmoscope, it has to be kept in mind that, as long as the pupil is fully occupied by light, from a surface of uniform luminosity, reflected into the eye, it does not matter whether the rays are condensed so as to enter the eye as a convergent bundle from a concave mirror or are caused to diverge from a plane mirror. The differences in the amount of light entering the eye in the two cases correspond to differences in the size of the image which it forms of the luminous surface, but not to any difference in the intensity of illumination of the unit of surface.

Take, in the first instance, the case of the reflection of parallel rays proceeding from a point of a distant evenly-illuminated surface. If we denote by s the area of the section of the bundle of parallel rays reflected from the mirror, and by p the area of the pupil, the amount of the light entering the eye is to that reflected as $p:s$, or calling I the amount of the reflected light, and i that of the light entering the eye,

$$i = \mathrm{I} \cdot \frac{p}{s}.$$

These rays focussed on the retina of an emmetropic eye will be brought to a point. This point, therefore, will have an intensity of illumination equal to that of the whole pupil. In myopia, or hypermetropia, instead of a point of illumination, there will be an area of diffusion of the light. The sum of the intensities of every point in that area will equal the intensity of the point at which rays meet, *i.e.* again, the intensity of the pupil.

In the case, then, of the image in the eye of one point only, there would be a difference in the degree of brightness of the area illuminated according as the image was accurately focussed on the retina or not. But if, instead of considering merely one point, we take the case of the reflection of rays from every point of a continuous evenly-illuminated surface, then it is evident that the retinal image corresponding to all points of that surface from which rays fill the pupil, is equally bright whether the eye is emmetropic or not. The diffusion circles in the case of ametropia will be so superposed that the illumination will be uniform and equal, for every point, to that of the properly focussed retinal image. For this reason an ametrope sees the sky of the same brightness as an emmetrope, provided only that his pupil is the same size as that of the emmetrope.

Not only is this the case, but it is impossible in any way to increase the brightness of the retinal image of the sky or any uniformly-illuminated surface. If, instead of being parallel, the rays entering the pupil from any point of the surface are divergent or are rendered convergent by means of a lens, a correspondingly greater number will enter it. The illumination of the pupil will, therefore, be greater the nearer the point from which the rays diverge, or to which they converge. The same will be the case for the illumination of the pupil from all points of the surface. The illumination will be inversely as the square of the distance of the luminous surface from the pupil. But the apparent size of any portion of the surface also varies inversely as the square of its distance from the eye, so that a compensation takes place. The visible brightness of an object is, therefore, not altered by altering the distance from which it is seen directly.

Next, to take the case where the object is not seen directly, but indirectly, through its image formed by a mirror or lens. We have then

I for the intensity of illumination of the unit of surface of the image, O^1 (if we disregard the loss of light on refraction or reflection). Putting L for the aperture of the mirror or lens, and J for the intensity of the object O at distance D

$$I = J \frac{O}{O^1} \cdot \frac{L}{D^2}. \qquad (a)$$

As the size of the object is to that of the image as the squares of their respective distances from the mirror or lens, this may be written $I = J \frac{L}{D_1^2}$, if D_1 = distance of image.

The same will be the case for the illumination of the pupil from all points of the surface. The illumination will be inversely as the square of the distance of the luminous surface.

The intensity of the image O^1 is therefore directly proportional to the aperture of the mirror or lens. That aperture is not, however, necessarily equal to the section of the bundle of rays entering the pupil from the image. If we call the area of the pupil as before *p*, and its distance from the image *d*, we get the size *l* of the portion of the reflecting or refracting surface from which rays from every point of the image enter the pupil, $l = p . \frac{D_1^2}{d^2}$, and if this value of *l* be put for L in (*a*) the apparent brightness to the image of the eye is $I = J \frac{p}{d^2}$.

But this is the same brightness as if the object itself were viewed directly at the same distance *d*, and consequently, also, the same brightness as at whatever distance it is seen directly. It follows also, for the same reason as where the pupil is filled with parallel rays, that it is immaterial, when we have to do with a continuous and evenly-illuminated surface, whether the eye is focussed for the image of the surface, formed by the mirror or lens, or not.

In the usual ophthalmoscopic examination, the source from which the illumination of the fundus is derived is not a continuous and evenly-illuminated surface of light. The flame in ordinary use is brighter towards its margins than at its centre. With a concave mirror the light is more evenly distributed over a larger area of the retina, and thus a more suitable illumination is obtained. The image of the flame then lies far in front of the retina, so that the diffusion circles from each point on the retina are large. Those from the borders of the image of the flame, by overlapping those from its more central parts, help to brighten the central area of illumination, and at the same time to produce a considerable illumination over the ill-defined margin.

We have seen that when the pupil is filled with rays the intensity of illumination depends upon its size. When, on the other hand, it is not filled by rays, the intensity will be as the proportion of the area occupied by rays is to the whole area of the pupil. In the illumination of the fundus by the ophthalmoscope mirrors in ordinary use, the reflected rays do not completely fill the pupil, as more or less shadow and penumbra is thrown on it by the central aperture. More of the pupil is, however, filled by rays reflected from a perforated concave mirror than when a plane mirror is used. This, then, is the main reason why a better illumination is got with a concave mirror. Other conditions being equal, we may make the central aperture in a concave mirror larger than in a plane mirror, and yet obtain the same illumination.

With a larger aperture more rays enter the observer's pupil, and consequently the apparent illumination is greater. An increase in the size of the aperture, while it increases the apparent illumination, at the same time diminishes the actual illumination. It follows, therefore, that the illumination obtained by the observer must be a maximum for some definite size of the aperture, which will vary according to the shape of the mirror, the size of the patient's pupil, and the distance of the mirror from the flame and the eye.

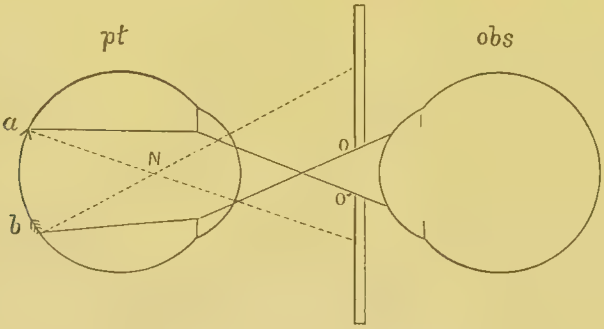

Fig. 156.

If I be the intensity of illumination corresponding to a full occupation of the pupil of area p with rays, and η be the area of the pupil not traversed by rays, then the intensity of illumination is, $I\frac{p-\eta}{p}$.

If e be the area of the central aperture in the mirror; F, the far point of the eye or the distance for which it is accommodated; d, the distance separating the mirror from the patient's pupil, then the section of the bundle entering the observer's eye from the patient's is, $e.p.\frac{(F-d)^2}{F^2}$.

The apparent brightness to the observer[1] (the area of whose pupil also $=p$) of an illuminated point on the observer's retina is then

$$I\frac{p-\eta}{p}.e.p.\frac{(F-d)^2}{p\,F^2} = I\left(1-\frac{\eta}{p}\right)\frac{e}{p}\cdot\frac{(F-d)^2}{F^2}.$$

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[1] Cf. Helmholtz, *Phys. Opt.*, new edition, p. 223.

The ophthalmoscopic field of vision, or the amount of fundus visible at one time on direct examination, depends upon the size of the pupil of the eye observed, upon the size of the aperture in the mirror of the ophthalmoscope and the inclination of the mirror, upon the distance separating the observer's eye from the eye observed, and upon the state of refraction. This is seen in Fig. 156. The visible area is greater the larger the patient's pupil and the larger the aperture in the ophthalmoscope, the nearer the distance separating the eyes of observer and patient and the more highly refracting the eye.

The amount of fundus visible on indirect examination is, other things being equal, inversely as the magnification. It increases with the degree of dilatation of the pupil of the eye examined, with the distance separating the observer from the convex lens, and with the size of the aperture in the ophthalmoscope.

This is seen in Fig. 157. The figure shows the size U of half the diameter of the portion of the fundus which can be seen by an observer whose eye is behind the ophthalmoscope aperture, half the diameter of which is represented by E at a distance d from the image formed by a lens of focal distance f, held at a distance D from the centre of the pupil of the eye, and through a pupil half the diameter of which is p. The case represented is that of emmetropia. It is supposed, too, that the lens is held at such a distance as to prevent the image of the iris masking that of the fundus.

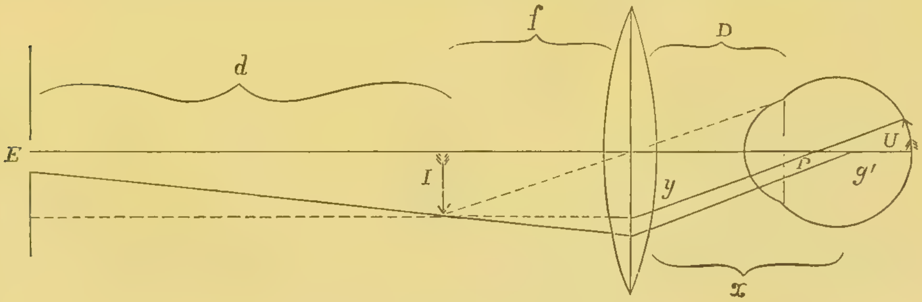

FIG. 157.

From the figure we have

$$\frac{I}{f} = \frac{U}{g'} \qquad \therefore \frac{fU}{g'} = I \qquad \text{and}$$

$$\frac{y - I}{f} = \frac{I - E}{d} \qquad \therefore y = \frac{f(I - E)}{d} + I.$$

Putting in the above value of I—

$$y = \frac{f\left(\frac{fU}{g'} - E\right)}{d} + \frac{fU}{g'} \quad . \quad . \quad . \quad . \quad a.$$

Again

$$\frac{y}{g'}=\frac{\mathrm{U}}{g'} \qquad \therefore\ x=\frac{g'\,y}{\mathrm{U}}$$

And

$$\frac{\mathrm{U}}{g'}=\frac{p}{x-\mathrm{D}} \quad \therefore \quad \mathrm{U}=\frac{g'\,p}{x-\mathrm{D}}.$$

Putting in the above value of x and simplifying, we get

$$\mathrm{U}=\frac{g'\,(y-p)}{\mathrm{D}}$$

Substituting for y its value at a.)—

$$\mathrm{U}=\frac{g'\,(d\,p+f\,\mathrm{E})}{f^2+d\,(f-d)}.$$

U therefore increases at the same time as g', p, and E, and diminishes with the increase of f. It also increases with D and with d.

When $f=\mathrm{D}$, a convenient position, and one in which the image of the pupil does not get in the way of the fundus—

$$\mathrm{U}=\frac{g'\,(d\,p+f\,\mathrm{E})}{f^2}.$$

As an example take

$d=250$ mm.; $f=70$ mm.;
$p=\ \ 3$ mm.; $\mathrm{E}=1{\cdot}5$ mm.; and $g'=15$ mm.

The amount of visible fundus is then approximately

$$\frac{15\,(750+105)}{4900}\times 2=5{\cdot}24 \text{ mm.}$$

that is, a portion the diameter of which is about three times as much as the disc. In the case of ametropia, as the magnification varies with the distance of the lens from the eye, the amount of the fundus visible at once is more or less than in emmetropia (with the same size of pupil and the observer's eye at the same distance from the aërial image), according as the magnification is less or greater than that of the emmetropic image. The corresponding formulæ for myopia and hypermetropia are rather long, and show nothing further. They are therefore not given.

THE MAGNIFICATION OF THE OPHTHALMOSCOPIC IMAGE.

By *the indirect method* of examination an inverted image is formed of the back of an emmetropic eye at the principal focus of the convex lens, and therefore, whatever be the distance of the lens from the eye, always at the same distance from the lens. The stronger the lens, too, the nearer will this image lie to it. It is evident, too, from Fig. 158

that the size of the aërial image is to the object at the back of the eye as the focal distance of the lens is to the distance from the second nodal point to the retina. If we call this latter distance g', and the focus of the lens f, the magnification $\frac{I}{U} = \frac{f}{g'}$. The distance g' may be taken roughly = 15 millimetres. The magnification with a lens of 75 millimetres or 3 inches focus, such as is commonly used for the purpose, would therefore be about 5 diameters, that with a stronger one less, *e.g.*, with a 2½ inch focus not much more than 4 diameters.

The real magnification or the size of the image formed on the observer's retina will depend upon how far his eye is from the aërial image. If, for instance, the observer obtains a clear image of the disc of the eye examined when his eye is at a distance from the convex lens equal to three times its focus, then the diameter of that image is approximately equal to half the actual diameter of the disc. The observer therefore sees the disc with the same degree of distinctness as he sees any external object which gives a retinal image of ·8 or ·9 mm. (*i.e.*, half the diameter of disc).

On direct examination of an emmetropic eye by another emmetropic and similar eye, the size of the image formed in the observer's eye will be exactly the same as the object from which the rays proceed in the eye examined. This is evident from Fig. 127. To estimate the magnification, then, we have to consider that the definition thus obtained is similar to that of any other external object whose retinal image is the same size. Thus, if we take the diameter of the disc roughly as 1·75 millimetres, we have merely to calculate what size of object at any particular distance would give a retinal image of the diameter of 1·75. Taking the same value for g' as before (and neglecting therefore any change which is caused in its value by the necessary accommodation), the definition would be about the same as that obtained by a penny held at between 10 and 11 inches from the eye. The diameter of a penny is about 31 millimetres, so that we have 1·15 : 15 = 31 : 266. The magnification is then as 31 to 1·75, or roughly, 18 diameters, if we compare the ophthalmoscopic distinctness of the image with that which the naked eye would give of the disc at 266 millimetres from its first nodal point, *i.e.*, at about 10½ inches from the eye. The apparent size of the magnified disc or other

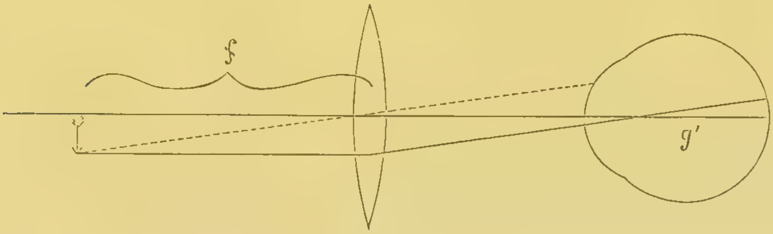

FIG. 158.

portion of the fundus of the eye depends on the distance to which it is mentally projected. To different observers the same disc will, on this account, appear magnified in different degrees.

As is explained in the chapter on refraction, and exhibited by Fig. 126, the magnification of the indirect ophthalmoscopic image in the case of ametropia depends on the distance at which the convex lens is held from the eye.

This may also be shown analytically in the following manner. We have, in the case of a lens in air where $f' = f$

$$\frac{obj}{Im} = \frac{u}{v}$$

And

$$\frac{u}{v} = \frac{u-f}{f} = \frac{obj}{Im.}$$

In myopia, where u is negative (as the object, viz., the image of the fundus oculi lies between the observer and the position given to the lens), this diminishes as u diminishes; but as the object remains always of the same size, the image formed of it by the lens increases in size as u diminishes, *i.e.*, as the lens is withdrawn from the eye. Until $u = o$, or the lens is withdrawn to the far point of the eye, the object is bigger than the image. From this onwards u is positive, and the image becomes larger and larger than the object, until $u = f$, when it is infinitely great, and a further withdrawal of the lens causes it to diminish in size, while at the same time, from being an erect image of an inverted image of the fundus, and consequently an *inverted* image of the fundus, it becomes an inverted image of this inverted image, or an *erect* image of the fundus. It is only, however, when the myopia is of extreme degree, and the lens used strong, that these successive changes occur within the range of withdrawal of the lens.

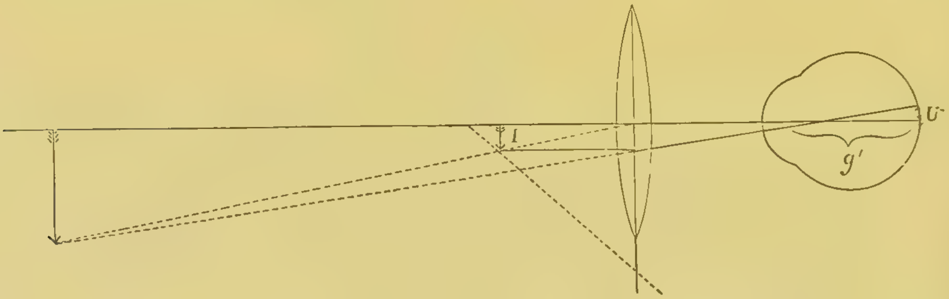

FIG. 159.

In hypermetropia u is positive, and increases as the lens is withdrawn from the eye, as the object is the erect image of the disc behind the eye. With the increase of u the relation of object to image gets less, which, as the object remains of the same sign, is tantamount to the image getting less.

We may get an approximate expression for the magnification in terms of the degree of ametropia, the focus of the convex lens, and its distance from the eye, in the following way:—In the case of myopia (see Fig. 159) put M for the distance of the far point from the nodal point of the eye. (To simplify we may look upon the two nodal points as merged into one.) We have then for the size of the image (β) at the far point compared with the object at the back of the eye, $\frac{\beta}{U} = \frac{M}{g'}$.

$$\therefore \beta = \frac{M\,U}{g'},$$

and for the relation of I (the image after refraction through the convex lens) $\frac{I}{\beta} = \frac{v}{u}$.

$$\therefore I = \frac{v}{u}\beta$$

Again, from the general formula 4 *b.*) $\frac{1}{u} - \frac{1}{v} = -\frac{1}{f}$

$$v = \frac{uf}{u+f}.$$

Putting then for u, $M - D$, where D equals the distance between the nodal point of the lens and the nodal point of the eye, and substituting the values found for β and v, we get—

$$\frac{I}{U} = \frac{f}{g'\left(1 + \frac{f-D}{M}\right)} = \frac{M}{g'\left(1 + \frac{M-D}{f}\right)}.$$

From both we see that I increases when D, the distance of the lens from the eye, is increased. From the first it is evident that I diminishes as M is diminished, that is, as the degree of myopia increases. From the second expression again, we find that by increasing f, that is, by making the lens weaker, I is increased in size. The equation also shows that when M becomes infinite, *i.e.*, when there is no myopia, $\frac{I}{U} = \frac{f}{g}$, as we have already seen is the expression for th magnification in emmetropia. But we evidently get the same expression for the magnification by making $f = D$, and yet the magnification is not the same as in emmetropia, but less, as the value of g' increases with the myopia. To distinguish between the g' in the case of emmetropia and that in myopia, we may write for g', g'_{M}, and the expression for the magnification in myopia, when the convex lens is held at the distance of its focus from the nodal point of the eye becomes $\frac{I}{U}=\frac{f}{g'_M}$, or if we put in the value of g'_M, which we get if the myopia, as is usually the case, is axial from the formula $\frac{G}{g}+\frac{G'}{g'}=1$, and put for G and G′, the distances respectively of the first and second nodal points from the foci, their equivalents, ϕ' and ϕ, and for g', M as before, this may be written

$$\frac{I}{U}=\frac{f}{\phi}\cdot\frac{M-\phi'}{M}$$

a value for I obviously less than $\frac{Uf}{\phi}$, the value in emmetropia, and the more so the smaller is M, that is, the higher the degree of myopia. The general formula in the case of axial myopia is then conveniently written—

$$\frac{I}{U}=\frac{f}{\phi}\cdot\frac{M-\phi'}{M+f-D}.$$

In the case of hypermetropia (see Fig. 160) we arrive by a similar

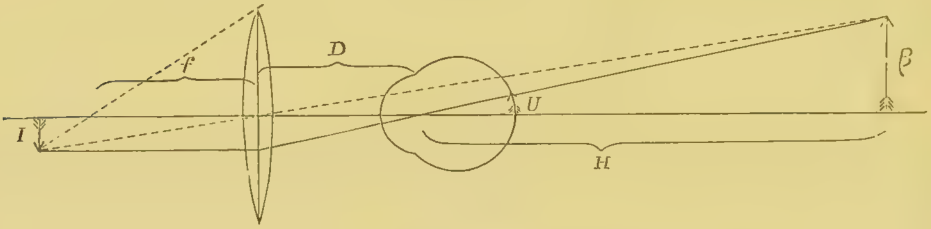

FIG. 160.

method, and calling the distance of the far point to the nodal point of the eye H, at the expression for magnification—

$$\frac{I}{U}=\frac{f}{g'\left(1-\frac{f-D}{H}\right)}=\frac{H}{g'\left(\frac{H+D}{f}-1\right)}.$$

As before, this becomes when $H=\infty$, that is when the hypermetropia is reduced to nothing, $\frac{I}{U}=\frac{f}{g'}$, the expression for the magnification in emmetropia.

From both expressions we see that as D, the distance of the lens from the eye, is increased, I diminishes. From the first we see that as H is diminished, that is, as the hypermetropia increases, I becomes greater.

And from the second it is evident that the diminution of f, or the increase in the strength of the convex lens, causes I to diminish.

For the case where $f = D$, or the lens, is held at the distance of its focus from the nodal point of the eye, we have as in myopia

$$\frac{I}{U} = \frac{f}{g'_H}$$

or by putting in the value of g'_H in the case of axial hypermetropia,

$$\frac{I}{U} = \frac{f}{\phi} \cdot \frac{H + \phi'}{H}$$

which shows that I is always greater than in emmetropia, and the more so the higher the degree of hypermetropia. The general formula in the case of axial hypermetropia for the magnification of the indirect ophthalmoscopic image is then

$$\frac{I}{U} = \frac{f}{\phi} \cdot \frac{H + \phi'}{H - f + D}.$$

To get the magnification in the case of ametropia by *the direct method* of ophthalmoscopic examination, we must first compare the size of the image in the observer's eye with the object in the eye examined, and then apply the same reasoning as for the magnification in emmetropia.

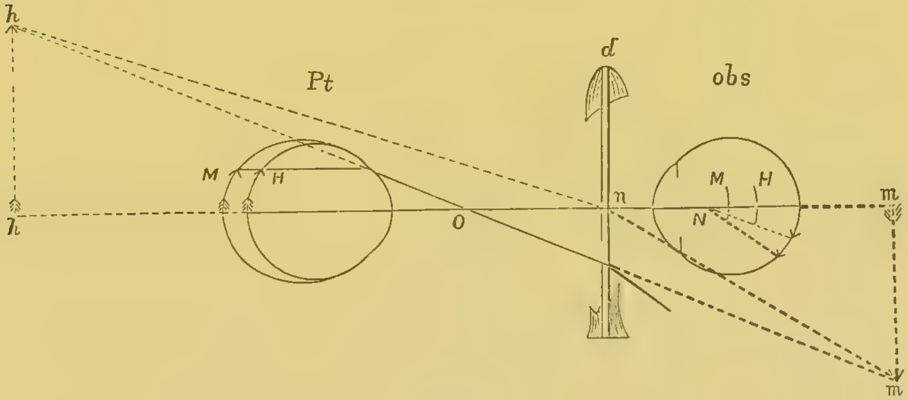

FIG. 161.

In Fig. 161 the difference between the magnification obtained in hypermetropia and myopia respectively, is shown for the ordinary position of the correcting lens, viz., further from the eye examined than its anterior focus. From the figure it is apparent that under these circumstances a larger image of any part of the back of the eye is obtained if it be myopic than if it be hypermetropic. The difference is greater, too, for equal degrees of ametropia the further the correcting lens lies from the anterior focus of the eye examined. The difference diminishes as the correcting lens is approached to that focal point. When the centre of the lens coincides with the anterior focus of the patient's eye, the magnification is the same for both myopia and hypermetropia, and the same as for ametropia.

If we denote by H and M respectively the distance to the far point of the patient's eye measured from its principal point, and by D the distance from that point occupied by the centre of the convex or concave connecting lens; by *Obj.* the size of the object at the back of the eye examined, and by *Im.* the size of the image on the retina of the observer's eye, while putting I for the size of the image of the same object at the far point of the patient's eye, we have for hypermetropia:—

$$\frac{Obj}{Im} = \frac{I}{H + \phi} \text{ and } \frac{Im}{g'} = \frac{I}{H + D}$$

for myopia—

$$\frac{Obj}{Im} = \frac{I}{M - \phi} \text{ and } \frac{Im}{g'} = \frac{I}{M - D}$$

from which we get for hypermetropia—

$$\frac{Im}{Obj} = \frac{H + \phi}{H + D} \cdot \frac{g'}{\phi}$$

and for myopia—

$$\frac{Im}{Obj} = \frac{M - \phi}{M - D} \cdot \frac{g'}{\phi}.$$

In axial ametropia we may put g' (the distance from the second nodal point of the observer's eye to his retina) equal ϕ (the distance from the anterior principal point of the patient's eye to his anterior focus). The formulæ then become respectively:—

$$\frac{Im}{Obj} = \frac{H + \phi}{H + D} \text{ and}$$

$$\frac{Im}{Obj} = \frac{M - \phi}{M - D}.$$

From these formulæ it is evident that the magnification is equal to emmetropia only when $D = \phi$. In hypermetropia it is less than in emmetropia, for $D > \phi$ (the usual case in practice) and less than in emmetropia, for $D < \phi$. The opposite holds good for myopia.

To take an example, a case of myopia fully corrected by a – 16·0 lens at the anterior focus of the eye, say 13 millimetres in front of the cornea, is examined with a – 18·0 behind the ophthalmoscopic mirror, and 20 millimetres in front of the cornea, what is the magnification compared with emmetropia? It is, obviously, as the focal distance of a 16·0 dioptre to that of an 18·0 dioptre lens, *i.e.*, as 9 to 8, so that if we take the magnification in emmetropia as 18 diameters, it would in this case be rather more than 20 diameters.

The relative depth of the different parts of the back of the eye seen with the ophthalmoscope can be estimated with tolerable accuracy by noting the number of the lens which the unaccommodated eye requires to obtain a clear definition, and allowing roughly 3 dioptres to 1 millimetre.

By the indirect examination differences in depth are rendered apparent by the parallactic displacement which takes place when the convex lens is given a slight movement from side to side. The structures lying nearer to the eye then appear to glide over the deeper-lying parts as their image moves more rapidly in the same direction as the lens.

Fig. 162 makes this apparent. In the figure H represents a nearer, and E a more distant point. At the same time the rays from H are drawn divergent as they emerge from the eye, and as if they came from a point *h* behind the eye, while those from E leave the eye parallel to each other. In the first position of the lens the image of this point is at H′. In the second (represented by the dotted lines) it is at H″. For the same movement of the lens the image of the point E moves from E′ to E″, and thus makes a smaller excursion.

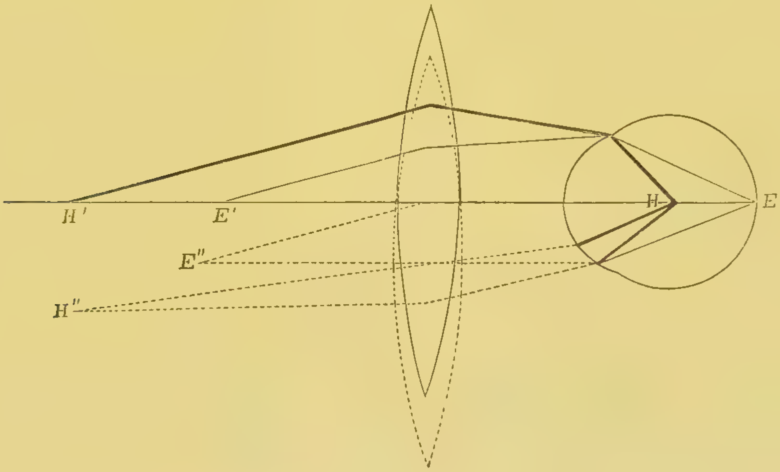

Fig. 162.

A fairly good stereoscopic view of the fundus is got with the binocular ophthalmoscope of Giraud-Teulon, which is so arranged, by means of two rhomboidal prisms, the angles of which are 45° and 135°, meeting at their acute angles behind and at the centre of the aperture of the mirror, that the aërial image can be seen with both eyes at the same time.

Many forms of ophthalmoscope are in use, differing from each other mainly in the kind of mirror used and the arrangements for bringing different lenses behind the aperture. For estimating refraction it is of advantage to be able to bring any lens behind the mirror without losing sight of the fundus, and this can be done with most modern instruments, but there is a tendency to multiply the number of lenses and introduce refinements in the mechanism of hanging the mirror, &c., which only add to the cost without in the least increasing the practical usefulness of the particular ophthalmoscope. The intervals between the lenses should not be less than one dioptre, and the higher ones may, without introducing any practical inaccuracy, be obtained by the combination of two lenses. In the examination by the direct method rays are only reflected into the eye from a very small portion of a plane mirror. If a strong illumination be required, it is therefore necessary to use a concave mirror with a short focus. Such mirrors have been introduced by Parent, and are fitted into some ophthalmoscopes in addition to the larger plane or less concave mirrors used for indirect examination.

CHAPTER XVII.

AFFECTIONS OF THE OCULO-MOTOR MUSCLES.

PHYSIOLOGICAL AND INTRODUCTORY.

THE eye is freely moved in all directions round a fixed point or *centre of rotation* by the action of one or more of its six external muscles. The centre of rotation lies about 1 millimetre behind the centre of the eye.

A consideration of the movements, as they actually occur, leads to the classification of the six muscles into three antagonistic pairs:—(1.) The lateral recti (externus and internus) which rotate the eye round a vertical axis: (2.) The superior and inferior recti which rotate the eye round an axis which meets the middle line in front of the eyes at an angle of 63°:

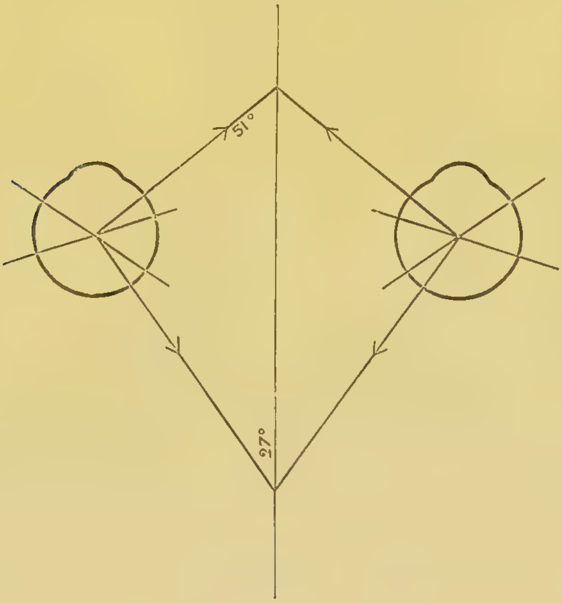

FIG. 163.

(3.) The two oblique muscles which rotate the eye round an axis which meets the middle line behind the eye at an angle of 39°—(see Fig. 163). The direction of action of the first pair is lateral—that of the second and third is represented by the figure.

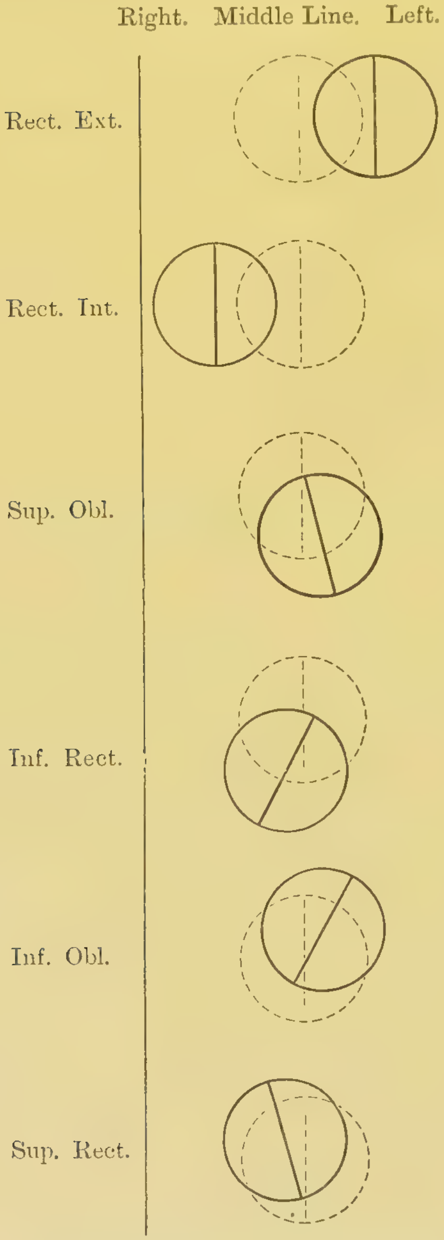

Fig. 164.—Diagram showing movement of cornea produced by each muscle separately.

If each muscle were to contract alone, the displacement of the cornea which it would produce would be as follows—(see Fig. 164). The externus would displace the cornea outwards without any torsion round the antero-posterior axis, the internus would displace the cornea inwards, also without any torsion. The superior rectus would displace the cornea upwards and slightly inwards, and at the same time twist the upper part towards the nose (medial torsion). The inferior rectus would displace the cornea downwards and slightly inwards, and twist the upper part away from the nose (lateral torsion). The superior oblique would displace the cornea downwards and slightly outwards, and producė at the same time medial torsion. The inferior oblique would displace the cornea upwards and slightly outwards, and produce lateral torsion.

Purely lateral movements are effected by the external and internal recti alone. Purely vertical movements are effected by the combined action of a straight and oblique elevator or depressor. Movements in intermediate directions are effected by the combined action of a lateral and two elevating or depressing muscles.

Purely vertical or purely lateral movements, starting from a position in which the visual axis is directed straight forwards in the horizontal plane (a position which may be called the initial position), bring the eye into what are called *primary positions.*

Movements of the eyes into primary positions take place without any torsion. In the case of purely lateral movements the reason of this is obvious, in the purely vertical it is because the torsional movements of the two elevators or depressors counteract each other. Movements into positions intermediate between purely lateral and purely vertical, or into what are called *secondary positions,* are accompanied by torsion, the direction and extent of which depends on the direction and extent of the lateral and vertical displacement from the initial position. When the eye is *abducted,* or turned away from the nose, the torsional effect of the oblique muscles preponderates, when it is *adducted, i.e.,* turned towards the nose, the torsional effect of the recti preponderates. It follows from this, what is found experimentally to be the case, that when both eyes are turned in the same direction into any secondary position, *i.e.,* upwards or downwards, to the right or left, the torsion is in the same direction in both eyes, as one is *ad*ducted the other *ab*ducted, so that in the one the torsion is lateral and in the other medial.

Two kinds of associated movements of the two eyes exist, and take place either singly or combined, viz.—(1.) movements in the same direction, and (2.) movements of convergence, or such as bring or tend to bring the two visual axes to cross each other in front of the eyes. A combination of these two movements takes place when any near object is fixed which is situated to one side of the sagittal plane. When, for instance, the object fixed is considerably to the right of that plane, and at the same time near the eyes, the right eye is turned outwards, while the left, being also directed to the right, is turned inwards. Further, the angular extent of the rotation of the left eye is greater than that of the right. Under these circumstances we must assume, as has been pointed out by Hering, that the internal rectus of each eye contracts, under the influence of an impulse to convergence, to an extent which would bring their axes to cross at a point in the middle line about the same distance from the eyes as the lateral point actually fixed, and that simultaneously the left internus, acting along with the right externus, under a stimulus towards a lateral associated movement, effect the remaining change in position which is required for the fixation of the object. The right eye is therefore kept in the position which it occupies by a contraction of the rectus externus, which to some extent is counteracted by a contraction of the internus, whereas the position of the left is the result of a contraction of the internus alone, effected, however, in response to two different impulses. If this view be correct, as there is good experimental evidence for supposing it to be, it follows that the same position of the eye may be associated with very different degrees of innervation, and consequent tension of its lateral muscles. This point is probably of no inconsiderable importance in connection with the etiology of strabismus.

Associated movements in the same direction are brought about by impulses, proceeding in all probability from definite centres in the brain. They are in great measure regulated by the desire for binocular vision, *i.e.*, for the fusion of the two retinal images into one, or what is often simply called, and will be afterwards spoken of as, *fusion.* This fusion only takes place as a rule when the two visual axes are both directed on the object on which attention is fixed. When only one visual axis is directed on the object looked at, whilst the other deviates in any direction, there is said to be a *strabismus* or *squint.* But even when the regulating influence of fusion is withdrawn, as happens when the one eye is occluded, the central impulse to associated movement in the same direction is sufficiently powerful to bring about more or less perfect correspondence in the degree of the change of position of the two eyes.

Associated movements to either side, or upwards and downwards, *i.e.*, movements into what are called the *primary* positions, are found to be less dependent on binocular vision than movements in intermediate directions, lateral and vertical combined, or the so-called *secondary* directions. In all probability this is because the more constant use of the eyes in the primary positions leads to a more perfect regulation of the centres by which they are actuated.

The greatest possible movement of the eyes takes place downwards, and the least upwards. The full extent of possible downward rotation is from 55° to 60°, that of upward rotation only about 30°. Not so much difference exists in the normal extent to which rotation inwards and outwards can take place, though the inward movement is slightly more extensive, or about 45°, the outward being rather more than 40°.

In the associated movements to convergence we recognise other regulating factors besides that of fusion, which is, however, undoubtedly the most important. Thus there is a more or less intimate connection between accommodative and convergent impulses, a connection which, in the form in which it exists in each individual case, there are many reasons for looking upon as partly inherited and partly acquired. Again, as when by occlusion of the one eye we remove the regulating influence of fusion, but yet do not find the convergence corresponding to that which might be expected from its connection with the accommodative effort required, even when we take into consideration the starting-point of convergence (or the position which the eyes assume before any convergent effort is made), we are forced to recognise still another factor influencing the centre for convergence. This factor has been called by Hansen Grut the consciousness of the distance of the object fixed. This consciousness is probably to a great extent an expression for the acquired regulation of the centres of association for convergence and accommodation, the persistence, in fact, of a state of innervation which, under existing conditions, is constantly called for.

The first factor, viz., fusion, is the more powerful in bringing about a perfect association in the convergent movements of both eyes, the greater the equality as well as acuity of their vision. Something also depends on the functional activity of the central mechanism of fusion. Thus we see, in the first place, when the vision of one eye has from some cause or other become impaired, a want of correspondence in the convergent movements in cases where such has previously existed only in a latent form; and, in the second place, it sometimes happens that when similar latent convergent effects have been always hitherto overcome in the interest of binocular vision, some debilitating influence leads all at once to a failure in the maintenance of this regulating power, notwithstanding the existence of good vision in both eyes. In other words, we may see a manifest squint occasioned by diminished fusion, due either to circumstances which obviously diminish the necessity for binocular vision, or to paresis of the nervous centre of fusion itself.

The power of fusion is much greater in the lateral than in the vertical movements, because much more frequently called for. In this respect, too, the internus has the advantage over the externus, while the power of all to overcome difficulties in the interest of binocular vision is strengthened by practice. As a rule, under normal conditions, when a distant object is fixed, a prism of 6° to 8°, held with its base inwards in front of one eye, the effect of which is to displace outwards the direction in which the object appears to lie, can be overcome, *i.e.*, the diplopia to which this gives rise can be removed by a movement outwards of the eye. This, however, is about the limit, whereas prisms with the bases outwards of 20° to 40° or more, may be overcome by an inward rotation. In a vertical direction any prism stronger than 4° (and often less) usually gives rise to diplopia which cannot be overcome. In exceptional cases a much greater vertical prismatic separation can be overcome by fusion.

A study of the conditions influencing associated movements not only throws light on the pathology of the ordinary varieties of strabismus, but also affords an explanation of many apparent irregularities in the manifestations of paralytic squints, and is consequently of diagnostic importance. The most intimate association between accommodation and convergence is present when a certain amount of accommodation induces at the same time a corresponding degree of convergence of the visual axes from their position of rest. What must therefore be looked upon as a theoretically normal connection is a simultaneous accommodation for, and convergence on, the same point. This could obviously only take place (as a result of the intimate association of the impulses to these two movements alone, and independently of other regulating factors) when there was at the same time emmetropia and parallelism of the axes of vision in the state of rest. Thus, in the case of emmetropia, when a point of $\frac{1}{4}$ metre from the eye is accommodated for, and therefore 4 dioptres of accommodation brought into play, there should be (provided the axes in the state of rest are parallel) convergence of the eyes on the same point, or what would now be called

4-metre angles of convergence. In order fully to understand, however, in how far the accommodative and convergent movements are ultimately connected in any individual case, it is obviously necessary to know in the first place the state of refraction of each eye, and in the second the position of equilibrium of the visual axes, or, in other words, the starting-point of convergence. If, for instance, in a case of emmetropia there is a divergent position of equilibrium, as is often the case, the ideal connection between the associated impulses would lead to a convergence less than towards the point accommodated for. If we suppose, as before, that accommodation took place for a point $\frac{1}{4}$ metre distant, the convergence, while all the time equalling 4-metre angles in amount, would direct the axes to a point further off than $\frac{1}{4}$ metre. Again, with hypermetropia, 2·0, as accommodation for a point $\frac{1}{4}$ metre from the eye, necessitates 6 dioptres of accommodation, the ideally associated degree of convergence of 6-metre angles would bring the visual axes from parallelism to be directed towards a point $\frac{1}{6}$ metre from the eyes, that is, the distance converged for would be nearer than that accommodated for. Where this connection existed on occlusion of the one eye, the other would be directed on the point for which it was accommodated, and the axis of the occluded eye would cross that of the fixing one at a nearer point, *i.e.*, the occluded eye would stand in the position of convergent strabismus. In a similar manner the ideal connection between accommodation and convergence would in the case of myopia lead to a divergent strabismus of the occluded eye.

The ideal connection between accommodation and convergence is far from being the rule. We meet in fact in all states of refraction with many cases in which there is both a latent defect and a latent excess in the degree of associated convergence. In emmetropia a near approximation to the ideal connection is the rule, although there is frequently some degree of latent divergence met with, less frequently latent convergence. In hypermetropia it is the rule to find the convergence lagging behind the accommodation in amount, so that there is only slight convergence, no deviation or even divergence found behind the occluding hand. In myopia, on the other hand, the amount of convergence is usually in excess of the accommodation, which leads to less divergence behind the occluding hand than might be expected, or to no deviation, or even in some cases to convergence.

The manner in which the relation between convergence and accommodation may be tested practically, is by causing the patient to fix with one eye an object at about 10 inches or ¼ metre from the eye, while the observer's hand is held in front of the other eye; on then removing the hand after twenty or thirty seconds, the previously occluded eye either makes a movement inwards or outwards, or remains in the position it assumed behind the occluding hand. If after removing the hand a movement of the eye takes place inwards, it shows that it was diverging, if outwards, converging relatively to the direction it should have. It is important in making this test to make sure that the uncovered eye is accurately accommodated for the object fixed. We may then proceed to determine whether the state of matters met with is accounted for by the position of equilibrium of innervation of the muscles, a test which cannot always be very satisfactorily made. What we desire to discover is the position of innervation equilibrium, independent of any accommodative impulse. In the case of emmetropia and myopia all that is required is to examine in the same way

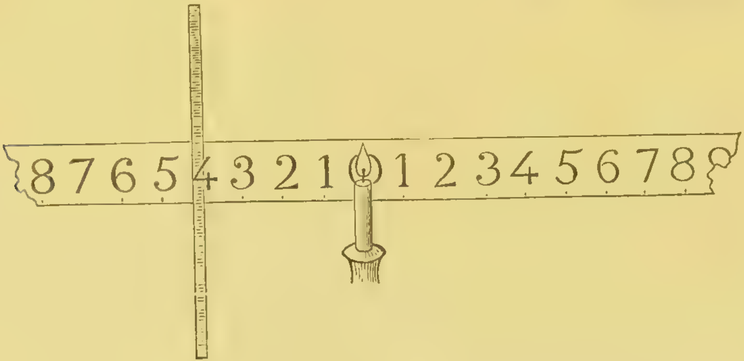

Fig. 165.

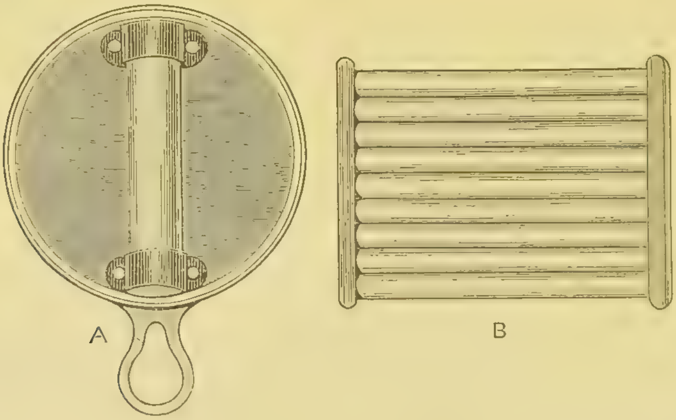

Fig. 166.

as first described, with this difference only, that instead of fixing a near object the uncovered eye is caused to fix a distant one. When there is hypermetropia an approach to the actual condition may be made, greater or less according to circumstances, by correcting the hypermetropia before making the test; but even when the total hypermetropia is corrected after the use of atropine, there may still remain a certain degree of convergence brought into play by an *impulse* to accommodation.

The method above described for determining the relative positions of the eyes is sufficiently accurate for practical purposes, so far, at all events, as the lateral muscles are concerned, as it is only in the case of the more pronounced deviations that any treatment is called for. It is sometimes desirable, however, to make more precise and more definitely quantitative measurements of latent deviations, and to do this, on distant fixation, a very simple and easily applied test is that with the Maddox glass rod and tangent scale. The patient stands facing a large scale, graduated in tenths of a metre angle for the distance (5 or 6 metres) at which the test is made. At the centre of the scale is a candle flame. This is fixed by the one eye. In front of the other a cylindrical rod of 5 to 6 mm. in diameter, of clear red glass, is held horizontally. The effect of this is to present to that eye a long vertical red line image of the candle flame. If there be no latent lateral deviation of the eyes, this linear image appears to pass through the centre of the flame. The existence of a lateral deviation, on the other hand, causes the line of light to appear to cut the scale at some distance, which can be easily read on the scale, to one side or other of the flame. Fig. 166 A shows the rod fixed opposite a slit in a small metal screen which prevents any object being seen by the eye in front of which it is held, except the elongated image of the flame. Fig. 166 B shows another arrangement consisting of a number of rods by which the same effect is obtained without any difficulty in bringing the cylinder opposite the pupil. Fig. 165 shows the manner in which the line of light is projected on the tangent scale.

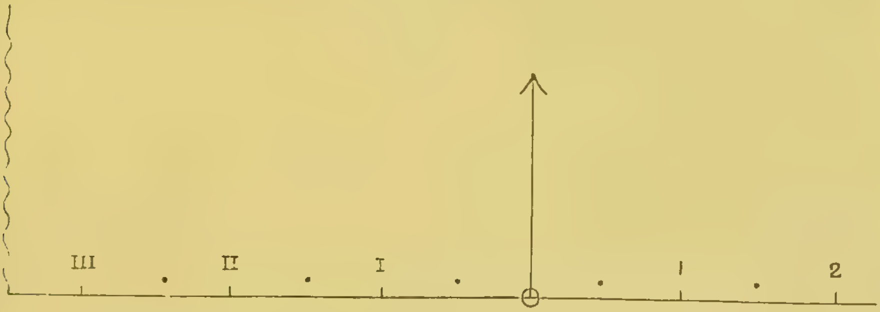

FIG. 167.

For the accurate measurement of deviations at nearer distances of fixation, the method introduced by Maddox, with a tangent scale and a prism of 10°, placed base upwards in front of the non-fixing eye, may be most conveniently adopted. Fig. 167 shows a portion of a scale, graduated in metre angles for a fixation distance of $\frac{1}{4}$ of a metre—a distance corresponding sufficiently to the ordinary reading distance—when the scale is held in front of the patient at the proper distance, and the central or zero point fixed, the lower arrow, *i.e.*, the false image caused by the prism, points to the number on the scale which indicates the latent deviation for that distance of fixation and to either side of zero, according as the deviation is one of convergence or of divergence. Each scale should have attached to it a string of such a length that when the free end is held by the finger of the individual examined against his own temple, the distance at which the scale should be used is obtained by keeping it stretched. The line of small print below the scale is introduced with the object of securing accurate accommodation, a matter of the utmost importance in making measurements from which any deductions as to existing relations between accommodation and convergence are to be drawn.

For measuring vertical deviations on distant fixation the glass rod and a vertical scale graduated in degrees, with a small flame at the zero point, is most convenient. The metre-angle scale has, of course, only a *raison d'être* in connection with lateral deviations.

PARALYSIS OF OCULAR MUSCLES.

When the action of any muscle of the eye is weakened there is a tendency for that eye to lag behind when an attempt is made to turn both eyes in the direction of action of the weakened muscle. In the case of paralysis of either of the lateral recti, the defect in the associated movements of the two eyes to the one side is often quite appreciable on mere inspection, it being evident where the weakness lies, though, if there be only slight paresis, such defect may, although it does exist, escape observation. In such cases, and mostly in the case of paresis of other of the oculo-motor muscles, the diagnosis is most readily, or it may be only, arrived at, by a consideration of the *diplopia* or double vision to which the condition gives rise. If in any position of fixation the two visual axes do not cross on the object fixed, there is, provided there existed previously true binocular vision, diplopia for that position. The diplopia is more and more marked, *i.e.*, the distance of the double images apart is greater and greater, the further the object fixed lies towards the boundary of action of the weakened muscle.

To facilitate the examination of the nature of the diplopia in any case, it is customary to place a coloured glass—most conveniently a dark red glass—in front of one eye, and cause the patient to look with both eyes at a candle flame held in the observer's hand at a distance of a few feet from the patient. This plan not only renders the diplopia more apparent to the patient, owing to the difference in colour of the two images which it produces, but also indicates to the observer, from the statements of the patient as to the relative positions of the coloured and uncoloured images, the relative positions of the image of either eye, from which he deduces the relative position of the eyes themselves, and consequently arrives at the conclusion as to which muscle or muscles fail to perform their function. The coloured glass should be held in front of the fixing eye, which is at the same time generally, though not always, the sound one, so far as the muscular defect is concerned. The image in the fixing eye being a central one, *i.e.*, one falling on the central area of the retina or the fovea centralis, is always more distinct than the more peripheral image in the deviating eye, consequently there is produced when the coloured glass is held in front of the fixing eye a less considerable difference in the strength of the two impressions than would be the case if it were held in front of the deviating one, and on this account there is less tendency to suppress mentally the image of one eye which would render the examination of the diplopia impossible.

If one eye be directed straight forward, all objects to the outer side of that fixed by it have their corresponding images on the inner side of the retina, while objects to the nasal side form images on the outer side of the retina. In order to be able to see these objects distinctly, we should have to move the eye outwards or inwards respectively, and to an extent varying with the position of each object. We gradually and unconsciously acquire the knowledge of the relative position of objects from learning, without being aware of it, what amount of innervation is necessary to effect and regulate this directing of the visual axes. We are accordingly in the habit of mentally projecting images lying on the nasal side of the centre of the retina laterally, *i.e.*, in the case of the right eye, to the right of the object fixed by the macula, and in the case of the left eye, to the left, and of projecting images lying to the temporal side of the centre of the retina medially, *i.e.*, in the case of the right eye, to the left, and in case of the left eye, to the right. At the same time, and for a similar reason—viz., as the result of the acquired knowledge of the normal position of the eyes—the fields of vision, whatever be their relative position, are as a rule mentally superposed, consequently the visual axis of a deviating eye is mentally projected so as to meet that of the fixing eye on the object fixed.

From these two laws it follows that when the position of the deviating eye is one of too great convergence, the image on it of the object fixed by the other eye, falling to the nasal side of the retina, is projected so as to appear displaced, with respect to the image of the fixing eye, in the opposite direction to that of deviation: therefore, in the case of too great convergence of the right eye, to the right, and of the left, to the left. This form of diplopia is called *homonymous*, as the image to the right corresponds to the right eye, and that to the left to the left eye. Again, if the deviation be one of too great divergence, the eccentric image is for a similar reason projected as before in a direction opposite to that of the deviation, which gives rise in this case to what is called *crossed diplopia*, as the image of the right eye appears to the left, and that of the left to the right. Therefore, relatively, too great divergence with respect to the object fixed gives rise to crossed diplopia.

Where there is a paralysis or paresis of any of the muscles which turn the eye outwards, there will be, for certain positions of fixation at any rate, relatively too great convergence, and consequently, where the other conditions favouring double vision exist, the diplopia will be homonymous. The pareses in which we might therefore expect to find homonymous diplopia are those of the external rectus, and also of either oblique muscle.

Again, when there is paralysis or paresis of any of the muscles which move the eye inwards, there is for certain positions of fixation relatively too great divergence, and consequently the existing double vision is crossed. The pareses in which we find crossed diplopia are therefore those of the internal, superior and inferior recti, muscles.

Besides lateral displacement of the double images, two other directions of displacement require attention, viz., the vertical and the torsional displacements. When one eye lags behind in an attempt at associated vertical movement, or, in other words, when its visual axis deviates from the point fixed, either upwards or downwards, the image on it of the point which is fixed by the fovea of the properly directed eye appears displaced in space in a direction, with respect to the image of the properly directed eye, opposite to that of the deviation—*i.e.*, when the deviation is downwards, the image of the deviating eye appears higher than that of the other; and when the deviation is upwards, the image of the deviating eye is lower than the other. To the lower eye corresponds, then, always the higher image, and *vice versâ*.

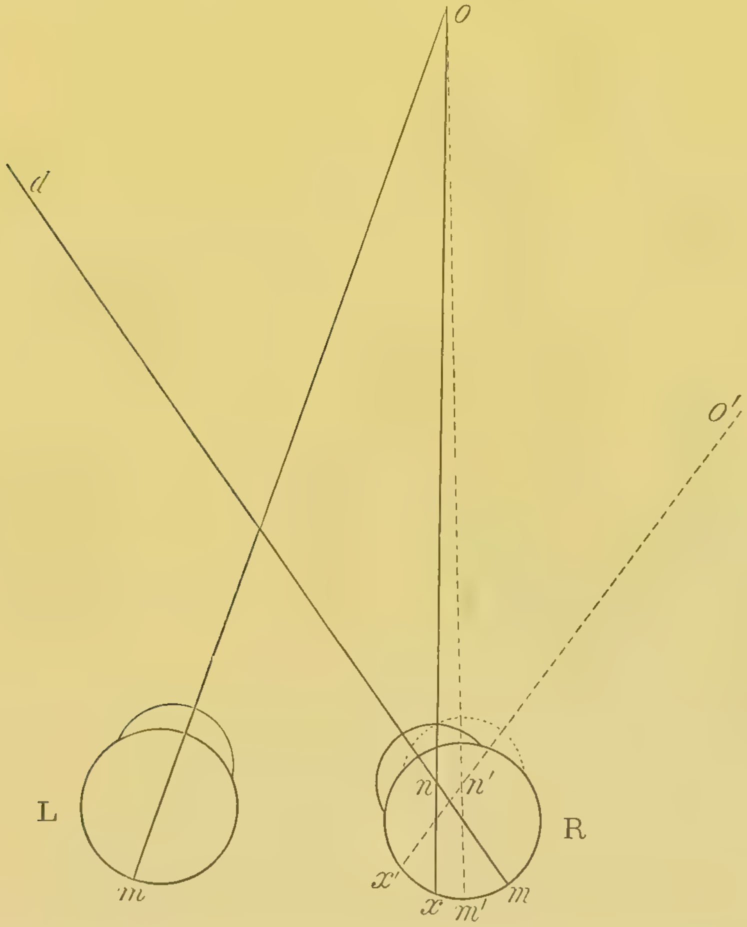

Fig. 168.—Shows manner of projection in abnormal convergence of right eye. *O*, point fixed by left eye; *m n d*, direction of visual axis of right eye, *m*, its fovea, *n*, its nodal point; *x*, position of image of *O* in right eye; *m' n' O'*, position in which individual unconsciously supposes right eye to be; *x' n' O'*, direction in which right eye projects image of *O*; $m\,x = m'\,x'$.—(After Alf. Graefe.)

The explanation of this is exactly similar to that which has just been given in the case of lateral displacement. The individual is, so to speak, unconscious of the position of the deviating eye, which he imagines to be directed in accordance with the hitherto existing laws of association. He therefore projects its image, not in accordance with its actual position, but with the position into which the associated impulse should have brought it, had there been nothing preventing it from responding to that impulse.

The same misinterpretation of the actual position of the deviating eye explains also the torsional displacement of its projected image, which is found for definite positions of fixation in some forms of paresis. When, owing to the paralysis or paresis of a muscle, the torsional effect which its action should produce on the position of the eye is absent or incomplete, the

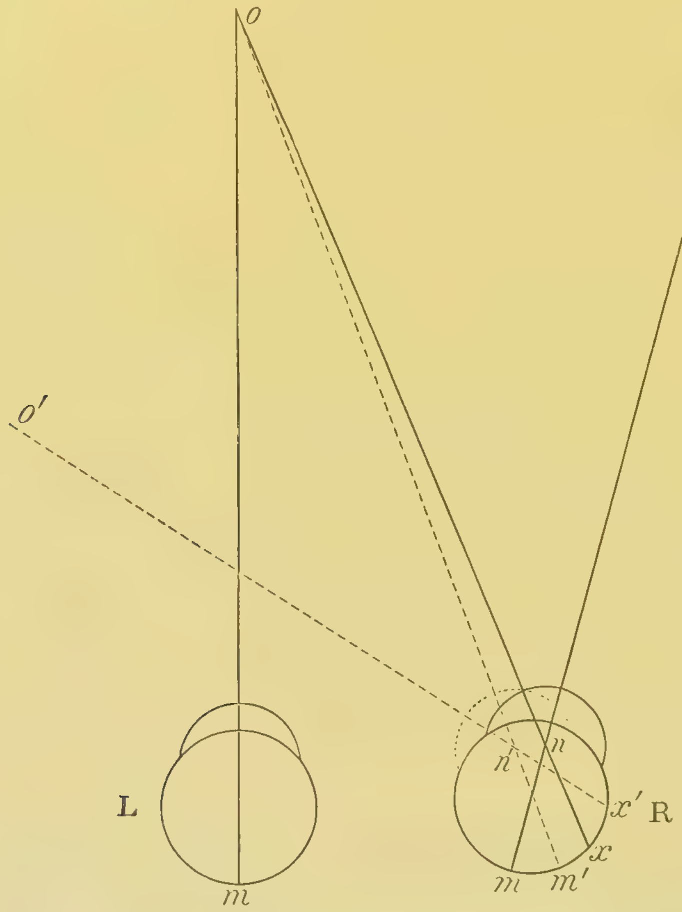

Fig. 169.—Shows manner of projection in abnormal divergence. Lettering as in Fig. 168. x' n' O', direction in which image of O is projected by right eye.

twisting of the projected image is also in the opposite direction of the deviation: thus if, instead of being twisted to the right, the eye remains untwisted, *i.e.*, remains behind the other to the left, as far as torsion goes, the projected image is twisted to the right.

The manner in which the image of the deviating eye is usually projected explains, too, a symptom which is often met with in fresh cases of paralysis or paresis, viz., a giddiness and uncertainty in the movements of the individual. If the patient be made to fix with the affected eye whilst the other is occluded, he sees everything to the side of the paralysed or paretic muscle displaced in the direction of action of that muscle. This may be readily demonstrated by causing him to grasp quickly with the hand of the same side at the object which he attempts to fix with the affected eye, when it is found that he grasps too far in the direction of action of the weakened muscle. In order that this experiment should succeed properly, his hand must have been previously out of view, and the attempt at grasping be made quickly, otherwise it is evident that the conditions for rectifying the error of projection are introduced. The giddiness disappears when the affected eye is occluded, but not if the sound one be occluded. For a similar reason it is evident that the difficulties introduced by paresis of the ocular muscles are greater when the affected eye is used for fixation. When this is the case, there is at the same time, when double vision exists, a greater separation of the double images than is the case when the sound eye is the fixing one, because the unwonted effort required by the affected eye for fixation of objects lying in the direction of the paretic muscle is associated with a greater movement in the same direction of the other eye, for which there exists no impediment to movement in that direction.

The deviation of the sound eye, when the affected one is used for fixation, is called the *secondary* deviation, as distinguished from the *primary* deviation of the affected eye when the sound one is used for fixation. The secondary deviation is evidently a measure of the degree of abnormal effort required by the weakened muscle to evolve the power required of it. The difference in the case of paralysis between the primary and secondary deviations constitutes one of the essential points of distinction between paralytic and ordinary or concomitant squint.

Two circumstances may affect the position of the double images to which paresis of a muscle gives rise: (1.) The previous position of equilibrium of the eyes; and (2.) the so-called *secondary contracture* of the antagonistic muscle. If the natural position of equilibrium be one of considerable divergence, the homonymous displacement of the double image in paralysis of an oblique muscle may not appear; if it be one of considerable convergence, on the other hand, the crossed character of the lateral displacement in paralysis of the superior or inferior rectus may not be present. When, again, there is a so-called secondary contracture of an antagonistic muscle, the area over which the diplopia becomes manifest in any case is extended, so that instead of merely existing in the direction of action of the weakened muscle, it extends over more or less of that of the antagonist as well. The occurrence of a secondary contracture is not altogether dependent on the degree of the primary defect, and may even exist when the original paresis has disappeared. When the secondary contracture is marked there is not so frequently diplopia. In the case of paralysis or paresis of the external rectus it is the internal rectus which is secondarily contracted, in paralysis and paresis of the superior oblique it may be either the inferior oblique, or superior rectus, or both.

Secondary contracture in ocular paralysis is not analogous to contracture of muscles elsewhere in the body. There is, for instance, no fixing of the eye in one position; it is free to move in the direction opposite to that of the supposed contraction. The term is obviously a misnomer. The cause of the preponderating action of the one muscle in any case is, in fact, the alteration produced in the relative innervation of the antagonistic muscles. The difference in the amount in different cases is no doubt owing to the different original states of innervation which exist.

It is not always easy to tell without determining the nature of the diplopia for which eye the paresis exists. This is, however, readily done by noting *to which eye that image belongs which lies furthest in the direction in which the separation of the two images is greatest.* That eye, therefore, lags behind, and does so owing to a weakness of the muscle which should move it in that direction.

The image which appears in the wrong place is often recognised by the patient himself as the *false* image, that which is seen in its proper place as the *true*. As the sound eye is generally used for fixation, the false image is at the same time most frequently less distinct than the true one, corresponding as it does to a peripheral retinal impression.

There may be paralysis or paresis of one or more ocular muscles without any diplopia. There are several reasons for this. When a deviation of the axis exists for some definite positions, and yet does not give rise to the diplopia which should characterise the strabismus, this is either because the image of the deviating eye is suppressed mentally, or because the patient has acquired the habit of projecting the images falling on the deviating eye in accordance with the position it assumes, and not in accordance, as is the rule, with the impulse to associated movements.

The first is the most common cause. But even when there is in no position any deviation, there may yet exist a paretic condition of one of the ocular muscles. Thus it often happens that the paresis is so slight that it can be overcome by fusion in the interest of binocular vision. This is, of course, most common in cases of paresis of one of the lateral muscles, as the power of fusion is greatest laterally. It is mostly met with in cases of internus paresis, as in such cases a much greater defect may be overcome in this way than is usually possible when the externus is weakened. In order to detect whether there is a hidden paresis of this nature, all that is necessary in the case of the lateral muscles is to place a prism with the base directed exactly upwards or downwards in front of one eye, and note whether there is (1.) any lateral displacement of the resulting double image in addition to the vertical displacement produced by the prism; and (2.) whether the lateral displacement increases or diminishes to either side. A more delicate test of the same nature can be made by using the Maddox rod instead of a prism. A lateral displacement which did not alter in extent to either side would, of course, not indicate a paresis of any particular muscle, but merely the position assumed by the eyes for fixation at the distance at which the test was made when deprived of the regulating influence of fusion.

Of the three nerves which supply the muscles of the eye, the third and sixth are more frequently paralysed than the fourth. If we consider the relative frequency of the occurrence of paralysis or paresis of isolated muscles, we find that that of the external rectus is by far the most frequent, while that of the inferior oblique is so rare that only a few cases have been recorded altogether. The superior oblique is also frequently paralysed, while isolated paralysis of the superior, inferior, and internal recti are amongst the uncommon affections of the eye.

Besides the isolated paralysis of the nerves which supply the muscles of the eye, or of the twigs supplying special muscles, we meet with all possible varieties of more complicated paralysis in one or both eyes, the diagnosis of which can be generally readily enough made from a consideration of the double images. An interesting form of complicated paralysis is the *paralysis of associated movements.* Thus we sometimes meet with more or less complete paralysis of convergence in cases where the two internal recti are nevertheless capable of acting in association with the externi. A considerable number of such cases have come under my own observation. On the other hand, a more or less complete loss of the power of moving both eyes to the right or left sometimes occurs when the convergent movements remain intact. Similar defects in associated movements in other directions are also met with, though less frequently. Occasionally there is paralysis of the same muscle or muscles on both sides. I have most frequently seen paralysis of both externi, but I have also met with paralysis of both interni, and of the two inferior recti alone. The latter condition is diagnosed from paralysis of associated movement downwards by the existence of diplopia in the secondary positions. Paralysis of all the external muscles of both eyes—what has been called by Hutchinson *ophthalmoplegia externa*—occurs either as one of the manifestations of gross cerebral disease, or—and then generally, so far as my experience goes, without any complication, owing to disease of the nuclei of third, fourth, and sixth nerves—sometimes the two third nerves alone are paralysed.

PARALYSIS OF THE EXTERNAL RECTUS, OR SIXTH NERVE.—When this condition exists the power of abduction is more or less completely lost. If there has previously been binocular vision, and the paralysis is recent, there is homonymous diplopia, which almost invariably extends more or less over to the side of the sound eye. The double images increase in distance apart the further the object is carried to the side of the affected eye. For equal lateral distances from the middle line, the double images are generally further apart for fixation below the horizontal plane through the eyes than for fixation above that plane. The reason of this is that divergence in the interest of fusion with the eyes lowered is more difficult, because rarely required, than with the eyes raised. At the same time the false image in the secondary positions is often slightly inclined from the true one above, and towards it below. This arises from the absence of torsion caused by the want of participation of the outward movement with the vertical movements of the eye.

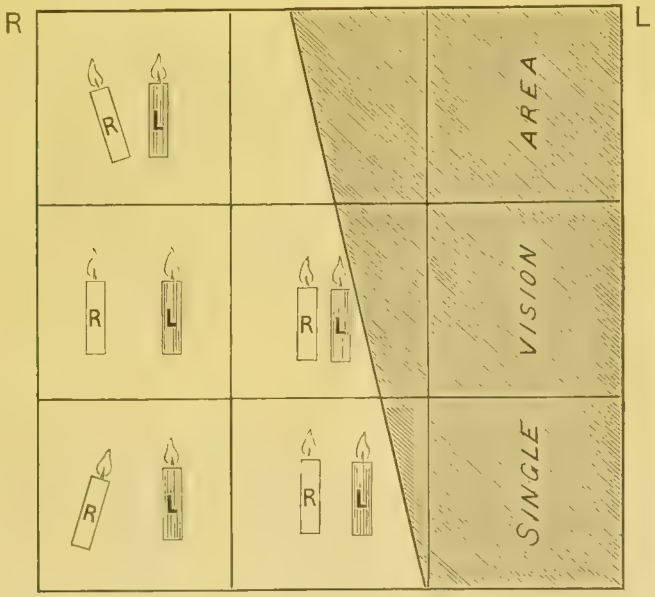

FIG. 170.—Position of double image in paralysis of right external rectus.

Fig. 170 gives schematically the positions of the double images. Paralysis of the right external rectus is more distressing than paralysis of the left, owing to its interfering more with reading. Frequently the patient keeps his head rotated to the side of the paralysed muscle, and fixes objects straight in front of him with his eyes turned to the other side. In this way he effects a more useful disposal of the area of single vision. The lesion producing paralysis of the sixth may be central or peripheral, and the causes various.

PARALYSIS OF THE SUPERIOR OBLIQUE MUSCLE, OR FOURTH NERVE.—In this paralysis there is more or less restriction in the power of moving the eye downwards, most marked when the eye is directed inwards. The diplopia exists only in the lower half of the field of fixation, unless the preponderance of the inferior oblique or superior rectus, or both, is asserted, as is often the case. The diplopia is homonymous, vertical, and torsional. The greatest vertical displacement occurs for fixation downwards and inwards; the greatest torsional displacement for fixation downwards and outwards; the images slope towards each other. Sometimes the homonymous character of the diplopia is not marked, or the images may even be crossed and sloping from each other. The explanation of this has already been given (see page 602). When there is preponderance of contraction of the inferior oblique, there is crossed diplopia in the upper half of the field of vision; when of the superior rectus, homonymous diplopia.

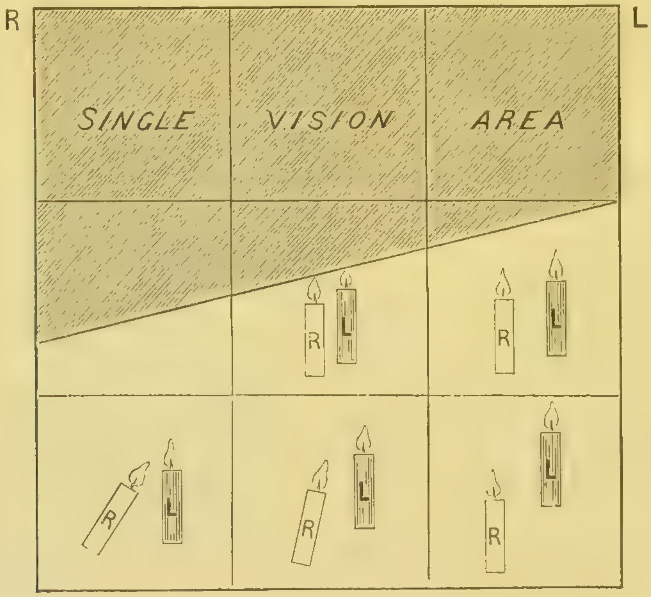

FIG. 171.—Position of double image in paralysis of right superior oblique.

Fig. 171 gives schematically the arrangement of the double images in the primary and secondary positions. (Compare with chart for inferior rectus.) Paralysis of the superior oblique causes difficulty in moving about, especially in going down stairs. The patient generally holds his head downwards, and towards the sound eye, and thus effects a more useful disposal of the area of single vision. The lesion in paralysis of the fourth nerve is either peripheral or central, and the causes various.

PARALYSIS OF THE INFERIOR RECTUS.—Movement of the eye downwards is more or less restricted, mostly when turned outwards. The diplopia exists only for the lower part of the field of fixation, except when there is a manifest preponderance of the superior rectus, or inferior oblique, or both, and is crossed, vertical, and torsional. The greatest vertical displacement is found for fixation downwards and outwards, the greatest torsional displacement for fixation downwards and inwards, and the double images slope towards each other. Occasionally the diplopia instead of being crossed is homonymous, and the images slope away from each other. When there is so-called secondary contraction the diplopia is less crossed, or even homonymous, if the superior rectus be contracted; markedly crossed if the contraction be mostly in the inferior oblique. Fig. 172 gives schematically the relative positions of the double image in the lower part of the field of fixation. (Compare with those of superior oblique.) The inferior rectus is seldom paralysed alone, though frequently along with other muscles supplied by the third nerve.

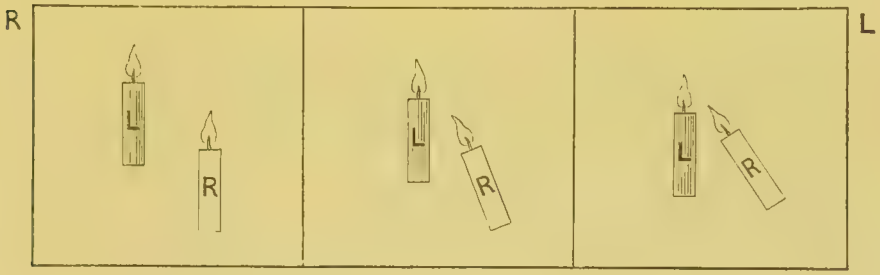

FIG. 172.—Paralysis of right inferior rectus.—Diplopia in lower portion of field.

PARALYSIS OF THE SUPERIOR RECTUS.—Movements of the eye upwards are more or less restricted, principally when it is turned outwards. Attempts to move the eye upwards are associated often with retraction of the upper lid, giving a staring appearance. The diplopia exists only for the upper part of the field of fixation, except when there is a manifest preponderance of the inferior rectus, or superior oblique, or both, and is crossed, vertical, and torsional. The greatest vertical separation of the images occurs for fixation upwards and outwards, the greatest torsional displacement for fixation upwards and inwards, and the images slope away from each other. When the eye is displaced in the direction of the antagonistic muscles, the diplopia, which extends to the lower part of the field of fixation, is then often homonymous. Fig. 173 shows the relative positions of the double image in the upper part of the field of fixation. (Compare with diagram for paralysis of inferior oblique.) The superior rectus is seldom paralysed alone, though often along with other muscles supplied by the third nerve.

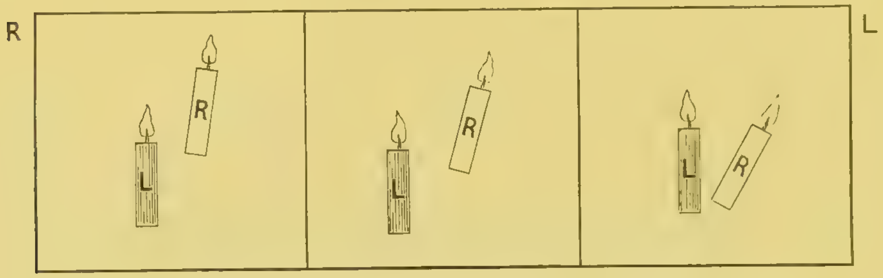

FIG. 173.—Paralysis of right superior rectus.—Diplopia in upper portion of the field.

PARALYSIS OF THE INFERIOR OBLIQUE.—Movements of the eye upwards are restricted, especially when it is at the same time adducted. Diplopia, unless there be a manifest preponderance of one of the muscles which rotate the eye downwards, only exists for the upper portion of the field of fixation, and is homonymous, vertical, and torsional. The greatest difference in height of the double images is seen for attempts at fixation

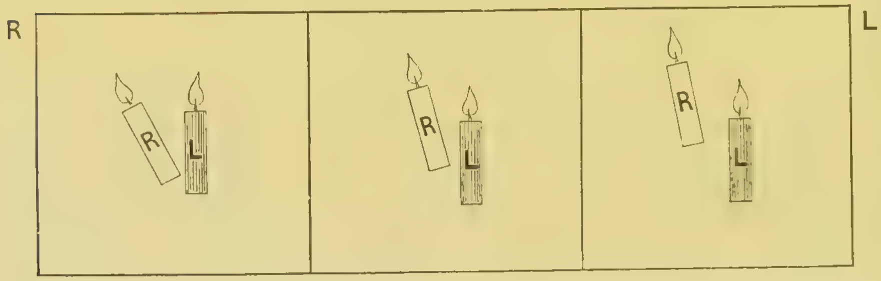

FIG. 174.—Paralysis of right inferior oblique.—Diplopia in upper portion of field.

upwards and inwards, the greatest torsion for fixation upwards and outwards, and the images slope away from each other. Fig. 174 shows the position of the double images in the upper portion of the field of fixation. (Compare with those caused by paralysis of superior rectus.) The inferior oblique is almost never paralysed alone, though often along with other muscles supplied by the third nerve. Occasionally a paralysis of this muscle has been met with, owing to the accidental detachment of its origin caused by wounds about the inner angle of the orbit.

PARALYSIS OF THE INTERNAL RECTUS.—There is more or less restriction of the inward movement of the eye. Diplopia, owing to manifest preponderance of the externus, stretches more or less over to the side of the field opposite to that of the affected eye. The diplopia is crossed, and the double images increase in distance apart as the object fixed is carried to the side of the affected eye. In the secondary positions there is slight sloping of the false image as well. Paralysis of the internal rectus is one of the rarest forms of isolated paralysis, though it is often the muscle most completely weakened when the third nerve as a whole is paralysed.

PARALYSIS OF THE THIRD NERVE.—There is more or less drooping of the upper lid, mydriasis and defective accommodation. Movements of the eye inwards, upwards, and downwards are more or less completely abolished. The amount of possible downward movement is not only dependent on the degree of paralysis of inferior rectus, but also on that of the internus as well. The more imperfect the adduction the less does the action of the superior oblique in turning the eye downwards come into play. Diplopia exists all over the field of fixation, the false image lies below the true, and is sloped outwards both for fixation outwards and inwards. Paralysis of the third nerve is very common, and is often incomplete, occasionally one, though more frequently two or more, of the muscles being alone affected, or more markedly affected, than the others. The lesion may be central or peripheral, and the causes various. When there is complete paralysis of the internus, it is not always quite easy to be sure of the action of the superior oblique, which is sometimes paralysed at the same time.

If, however, there be any appreciable torsion of the eye inwards when the attempt is made to look down, we may be pretty sure that the fourth nerve is not paralysed.

The lesions giving rise to ocular paralysis may be peripheral or central. Peripheral lesions may be situated within the orbit or affect the nerves in some part of their course within the cranial cavity. Central lesions, again, may be situated at the base of the brain, and directly or indirectly involve the nuclei of the third, fourth, and sixth nerves, which all lie within a space of about 1 inch long by ¾ inch broad, at the base of the fourth ventrical and aqueduct of Sylvius. Again, they may be situated in some parts of the brain above the nuclei, and involve fibres passing to them.

It is by no means always easy to differentiate in any particular case between a central and a peripheral lesion. The points to be considered in attempting a diagnosis of this nature are—(1.) the completeness of the paralysis, and (2.) the existence and nature of complications. An isolated and complete, or nearly complete, paralysis of the fourth or sixth nerve is almost certainly of peripheral origin; often, indeed, the cause lies in the orbit, but the existence of other symptoms, pointing to changes at the base of the brain would be strongly suggestive of its being intracranial. Paralysis of the third nerve presents greater difficulties, though in the main the more complete it is, if isolated, the more likely it is to be of peripheral origin. If associated with paralysis of the fourth and sixth of the same side, or of the opposite third, but unaccompanied by any other symptoms, the cause is almost certainly nuclear. In cases where paralysis of the fourth nerve on one side, and the sixth on the other, constitutes either the sole or the main symptom, there is great probability of the lesion being situated centrally with respect to the nuclei and to the side of the paralysed sixth, as the fibres of the fourth decussate, while those of the sixth do not. In every case the most important positive or negative information is to be got by ascertaining the condition of functional activity of the facial, sympathetic, and fifth nerves.

Of course in many cases of severe intracranial disease or injury the ocular paralyses are of subordinate importance to the other symptoms, and consequently the diagnosis of less significance.

To complete the diagnosis in any case, we should not only be able to fix on the site but also on the nature of the lesion, *i.e.*, whether the interruption in the function of the nerve is the result of neuritis, atrophy, pressure from hæmorrhage, or a tumour, or is more indirectly due to interference with its blood supply. It is only rarely that a complete diagnosis of this nature can be made. Syphilis is the cause of many cases, and the syphilitic lesion may be of every possible nature, and situated either centrally or peripherally, the latter being much the more common. There is a great tendency to recurrence in syphilitic cases, and the different attacks do not always involve the same nerves. Oculo-motor paralyses are also met with in locomotor ataxia and other degenerative spinal and basal affections.

Rheumatic paralysis, or that due to cold, is common, and possibly always peripheral, although I am not aware that any opportunities have occurred for studying their nature, as they are usually recovered from. Other less common causes are acute fevers and poisonings of the nerve centres, amongst which may be mentioned diphtheria, which, though frequently followed by paralysis of accommodation, seldom gives rise to oculo-motor paralysis. Occasionally we meet with congenital paralysis, the most common being ptosis and paralysis of one or both sixth nerves.

Many of these cases are no doubt due to congenital muscular defects, and are not true paralyses at all. Our knowledge on this subject is, so far, very defective.

The *treatment* of ocular paralysis should be general and local. When the primary cause of the paralysis, be it rheumatism, syphilis, or changes in the nervous system, or due to diphtheria or any other form of poisoning, is apparent, or when there exist sufficient grounds for suspecting any definite cause, the treatment found to be most efficacious in the particular affection should be at once adopted.

Local treatment may be instituted on three lines:—(1.) Passive movements of the eye in the direction of the action of the weakened muscle: (2.) Attempts to induce active movements of the weakened muscle: (3.) Operations calculated to bring about a more advantageous relative position of the paralysed muscle and its antagonist. Passive movements of the eye are made by seizing the conjunctiva with the fixation forceps and rotating the eye forcibly several times in succession, and once or twice daily in the direction of the action of the weakened muscle. This can be done without causing any pain by the use of cocaine. Attempts to induce active movement of the weakened muscle may be made in many ways. The simplest method is to cause the patient to fix binocularly some definite object, such as a printed word, and to carry this object gradually further and further in the direction of the area of double vision, whilst he is all the time making an effort to fuse the images of the two eyes which tend more and more to separate. The tendency to fusion is sometimes so strong in cases where the internal rectus is paretic, that the strain which maintaining binocular vision entails is not only the cause of considerable discomfort, and even pain, but may not improbably tend to retard the complete recovery. In such cases the patient may obtain great relief by the use of prisms of 3° or 4°, with their bases inwards in front of each eye. By this treatment the efforts to fusion are supported and the strain lessened.

Electrical stimulation of the weakened muscle, either by the constant or induced current, is recommended by many, and is occasionally of use. One pole is brought in contact with the forehead, and the other with the closed lid over the affected muscle. There is a great tendency for the paralysis of ocular muscles to disappear more or less suddenly, and after having existed for a longer or shorter period. Some cases, indeed, only last a few days. They are consequently well suited to confirm the faith which many place in electro-therapeutics, which in point of fact is about as successful in the case of ocular as in other paralyses.

Operative interference should not be thought of until the condition has remained unaltered for a sufficiently long period to render impossible any improvement by other means. Treatment by operation is only suitable in a small proportion of cases of paralysis of oculo-motor muscles. Interference of this nature should in the first place be deferred until a full year has passed since the onset of the paralysis. The advisability of operating will then depend upon whether the affection of innervation of the ocular muscle is the main or only symptom present, or whether it constitutes merely one symptom among many others indicative of serious cerebral disease. In the latter case nothing should be done; in the former, if one muscle alone be weakened, or if the paresis be only marked in the case of one muscle, it is sometimes possible to improve matters more or less considerably.

As isolated paralyses of the muscles supplied by the third nerve are of comparatively rare occurrence, it follows that the cases which offer a chance of improvement by operation are persistent cases of paralytic weakening of the external rectus and of the superior oblique. Cases of paresis of the external rectus of one eye are not only amongst the most frequent, but fortunately at the same time those for which most can be done by operation. In such cases the operative effect can be regulated with greater nicety, as, just as for concomitant convergent strabismus, the improvement in the relative position of the eyes can be produced by the combination of tenotomy of one or both internal recti, with the advancement of one or both external recti, according to the degree of the defect to be rectified, while there is besides a greater natural tendency, as has already been explained, to the fusion of the double images which frequently heightens the effect of a suitable operation.

The cases for which operations may be performed present considerable differences with respect to the completeness of the remaining paralysis as well as the degree of manifest preponderance in the action of the internal rectus or antagonist muscle; the extent of the tendency to fusion must also be taken into consideration in the selection of what operation or operations should be performed in any given case. The main object of any operation is to displace the area over which double vision exists as much as possible to the side of action of the paralysed muscle. In all cases, therefore, in which some power has been retained by or restored to the external rectus, it will generally be possible to effect this to an extent which is practically sufficient, by correcting the degree of convergent squint which exists for the fixation of objects in the middle line; while the improvement thus obtained will be more complete, the greater is the degree of such remaining power and the greater the desire for fusion. What in most cases should first be done is tenotomy of the internus of the other eye. If this is not sufficient, it may be followed, either at the same time or afterwards, by advancement of the paretic muscle and tenotomy of its antagonist. The worst cases may require in addition advancement of the external rectus of the sound side.

Cases of persistent paresis of the superior oblique do not lend themselves to such complete readjustment, as it is impossible either properly to advance the paretic muscle or to weaken the mechanism of rotation which is antagonistic to that produced by the superior oblique, as that could only be effected by tenotomy of both the inferior rectus and the inferior oblique, the latter of which cannot be satisfactorily done. Nevertheless, by tenotomy of the inferior rectus of the other eye, a very considerable displacement of the area of diplopia can be obtained in suitable cases, as was first pointed out by Alfred Graefe.

SPASMS OF OCULAR MUSCLES.

Spasmodic contractions of single oculo-motor muscles are not only of extremely rare occurrence, but are, owing to their inconstancy, very difficult to diagnose. A much more common occurrence is a spasm of associated movement. Thus, in consequence of cerebral irritation, to account for which there may or may not be some definite lesion, and which may be variously localised, a forcible deviation of the eyes takes place in some particular direction to either side, or upwards or downwards, or it may be a spasm of the convergent movement of the two eyes. At other times one meets, and mostly, in my experience, in cases of severe cerebral disease, with what has been called by Alfred Graefe a disjunction of the co-ordinated movements of the eyes, in which the movements of the eyes appear to be greatly or entirely independent of each other, and to be at the same time deprived of volitional control.

No general lines can be laid down for the treatment of such cases of associated or dissociated involuntary movements. Each case has to be considered for itself, but the important point to remember is that they are by no means invariably indicative of serious cerebral disorder.

CONCOMITANT STRABISMUS.

In the normal state of the eyes, when any object is looked at, the visual axes of both eyes are directed simultaneously on the same point of the object. There is, therefore, simultaneous *fixation* with both eyes. When only one eye is directed towards, or fixes the object engaging attention, while the line of vision of the other crosses that of the fixing eye, either in front of or behind, or above or below it, there is said to be squinting or strabismus. In the scientific acceptation of the term there can only be strabismus of one eye at a time, as it is clear that one eye must always, when it is possible at all, be directed on the object looked at.

Bilateral deviations and conditions of vision which render this impossible do not come under the category of squints.

According to the direction assumed by the axis of the deviating eye we have to distinguish between strabismus convergens and divergens, and strabismus sursum vergens and deorsum vergens. The vertical deviations, upwards and downwards, though often combined with lateral deviations, are rare alone, and are consequently of less practical importance. They frequently remain after the lateral deviations have been corrected by operation. Convergent and divergent squints are common.

When there is for no distance of fixation, either near or remote, a coincidence of the axes of vision, the strabismus is said to be *absolute*. When both eyes are properly directed on fixing a near object, but one axis misdirected when a distant object is looked at, or when the misdirection of one axis takes place only on fixation of a near, but not of a distant object, the strabismus is said to be *relative*.

The strabismus may be *constant* or *intermittent*. It may always be confined to the one eye, *i.e.*, it may be *mono-lateral*, or it may be *alternating* when objects to the one side are fixed by the one eye, and to the other side by the other eye. It may or may not be associated with diplopia.

As we have already seen, besides manifest strabismus, similar deviations are found to occur when one eye is occluded. There is then said to be a *latent* strabismus. The consideration of latent forms of strabismus is of importance, as they may under certain circumstances become manifest, or they may—though by no means so frequently as was at one time supposed—give rise to pain or discomfort, to that form of asthenopia which is called *muscular asthenopia*.

Mere inspection is not always sufficient to enable us to diagnose a squint, as on the one hand there may be a slight deviation without its being apparent, and on the other hand there may appear to be a deviation when in reality both eyes are properly directed. This may occur either when the eyes are not fixing the object which it is supposed they are fixing, or when the angle between the optic and visual axes is considerably larger or smaller than usual. Thus in hypermetropia an apparent divergence is due to an abnormal size of this angle, whilst in some myopes a negative value of the same angle gives rise to an apparent convergence which is sometimes extremely marked. It is necessary, therefore, in some cases at any rate, to adopt the following method of testing whether there is or is not any squint. The patient is made to fix some distant object; whilst he is doing so the observer covers one eye, and notices whether the one which remains uncovered makes any movement in any direction. The patient is then again made to fix the same object, and his other eye is in the same manner occluded by the observer's hand, and any movement of the uncovered eye looked for. A similar test is then made for the fixation of a near object—at 10″ from the eye. If the one eye has been misdirected while both were open, and if it at the same time is in possession of sufficient visual acuity for central fixation, it will take up the fixation of the object looked at as soon as the fixing eye is occluded from vision. In order to do so it will have to make a movement in a direction exactly opposite to that in which it previously deviated, so that a movement outwards of the other eye, on occlusion of the fixing one, is a proof of the existence of manifest convergent strabismus, a movement inwards, of manifest divergent strabismus. If the movements just described take place both for fixation of near and distant objects the manifest strabismus is diagnosed as absolute, if only for one or other, as relative.

Care must be taken that the occluding hand be not transferred too quickly from the one eye to the other, which would cause a difficulty in distinguishing which was the squinting eye. Time must be allowed for the fixation to take place in the way in which it is ordinarily effected. If the conditions of fixation be the same for both eyes, the squint is almost invariably transferred from the one eye to the other, *i.e.*, the occluded eye, whether it be the one usually employed in fixation or not, is misdirected. This can generally easily be seen by observing the position which it assumes behind the occluding hand. The fact is, that although the defect is one rendering the position of the two eyes either too convergent or too divergent relatively to the distance of the object looked at, it necessarily only becomes apparent on the one, as the other has to maintain a proper direction.

A truly alternating squint presents the following characteristics. If either eye, by first covering the other with the hand, be caused to fix an object in the middle line, *i.e.*, straight out from the nose, it subsequently remains for an indefinite period, after removing the occluding hand, in the position of fixation. It is evident that it is immaterial to the individual which eye he makes use of for fixation in this position. Again, on moving the object of fixation to the right, it is fixed by the left eye, whilst the right eye fixes it when situated to the left of the middle line. Both eyes have equal, or very nearly equal vision, and are often emmetropic.

The ordinary squint differs from the paralytic squint in this respect, that whereas in the latter the extent of the deviation increases in certain directions, it remains in the former the same for all directions. For this reason it is often called *concomitant.* The degree of concomitance, too, as measured by accurate tests is found to be astonishingly complete. Another difference between the two forms lies in what is called the secondary deviation. Whereas, as we have seen (page 601), the secondary deviation in the case of paralysis, or that deviation which takes place when the affected eye instead of the sound one is used for fixation, is greater than the primary deviation, the deviation in concomitant strabismus is the same when either eye fixes. In concomitant squint, as well as paralytic, there is often less convergence when the eyes are directed upwards, and more when they are directed downwards, than when fixation takes place for objects in the horizontal plane through the eyes.

In most cases of concomitant squint there is a distinct, though rarely considerable diminution in the extent to which the eye can be abducted if the squint be a convergent one, or adducted if it be divergent. At the same time there is usually a corresponding increase in the power of movement in the opposite direction. This restriction in the extent of lateral movement does not, however, correspond in amount to anything like the degree of squint. This fact is of importance in connection with the views which are by some entertained as to the etiology of strabismus.

One of the most frequent conditions met with in strabismus, the signification of which is discussed further on, is a greater or less degree of amblyopia of the squinting eye.

CONVERGENT CONCOMITANT STRABISMUS usually begins between the ages of one and four, though sometimes earlier, and not infrequently later. In a very large percentage of cases there is hypermetropia of one dioptre or more. There is usually no diplopia, though the existence or not of diplopia depends greatly on the age at which the squint first begins, and the degree of amblyopia of the squinting eye. In truly alternating convergent strabismus there is, however, no diplopia, although both eyes have generally nearly, if not equally, good vision. Sometimes the squint begins by being periodic and relative, *i.e.*, it occurs occasionally, and only when the child directs its attention on near objects. The periodic character of the squint may persist, or may sooner or later give place to a constant relative or absolute deviation.

A periodic squint sometimes recurs with remarkable regularity every other day, or at certain times every day, and when this is the case, the patient is more or less conscious of diplopia.

In cases where convergent squint is associated with myopia there is almost invariably diplopia. This is because it then begins later—seldom before, and generally considerably after, the tenth year. There has therefore previously been true binocular vision. For a long time, at least, it remains relative, existing only for a distance, there being proper binocular fixation for near objects. This is another reason why the patient continues to be conscious of the diplopia produced. Binocular fixation, besides being in many cases more valuable for objects lying near the eyes, is specially so in the case of myopia where distant objects are indistinct. Hence it is that the abnormal tendency to convergence existing in some cases of myopia is for long overcome during the fixation of near objects, remaining latent within a certain distance, though manifesting itself as a convergent strabismus on the fixation of any object beyond that distance. But the distance for which binocular vision is possible has a tendency, too, to become gradually less. Even, however, when in the course of time, owing to the gradual shortening of this distance, the relative has eventually become converted into an absolute convergent strabismus, the diplopia usually still remains.

The degree of faulty convergence varies greatly in different cases from an amount which is inappreciable, and can only be detected by a proper examination, to an extent in which the cornea is almost entirely buried behind the inner canthus. However excessive the squint is, there probably always remains some possibility of further adduction.

It is of importance to distinguish two elements in the squint, viz.—(1.) the *permanent* amount, and (2.) the *accommodative* addition. The permanent amount is that which still remains when no attempt at accommodation is made. The accommodative portion is a varying amount, dependent on the extent of the demand for accommodation. It is the degree of the permanent manifest squint alone which determines the extent of operative effect required.

Different methods may be employed for the measurement of the extent of deviation. One of the simplest, and one which is at the same time sufficiently accurate for practical purposes, is the linear measurement with the strabometer. This instrument is applied to the surface of the lower lid of the squinting eye, and the position of the millimetre scale noted, which is cut by the tangent to the outer margin of the cornea, whilst the other eye is fixing a distant object. The fixing eye is then covered, and the new position cut by the same tangent on the scale, whilst the squinting eye is used for fixation of the same distant object, is next observed. The distance between the two points thus found on the scale gives the linear deviation in millimetres. Many who take the trouble to make measurements at all prefer to estimate the amount of deviation in degrees. This is generally done by causing the patient to fix a point on a perimeter, and to observe how many degrees from that point it is necessary to carry a taper or candle, so that its reflected image shall occupy the centre of the cornea of the squinting eye. There is, I believe, no special advantage in obtaining an angular value for the amount of squint, although it is claimed that in this way measurements are obtained which are more strictly comparable than by means of the linear method, owing to differences in the size of the eye not being taken into account in the strabometer method.

The only scientific basis of measurement is that of the metre angle, as by such measurement we may ascertain in how far the associated movements of convergence and accommodation are mutually interdependent, or are in correspondence with the normal conditions.

The method of objective strabismometry, which I have used in a very large number of cases, with the object principally of measuring the effect of operations, is one which enables one, with a little practice, to attain an accurate measurement in metre angles without the expenditure of more than a few seconds' time. It is a modification of Priestley Smith's so-called "tape method," and, as now practised, is mainly due to Maddox. The metre angle scale referred to at p. 610 is made to serve the purpose of the objective as well as the subjective test. The method of using it for the former is thus described by Maddox:—"Place the patient opposite the candle, at the distance of one metre, as measured by a string of that length permanently suspended by one end from the centre of the scale. Let the observer's head be placed rather lower than, and in the same vertical plane as, the line between the flame and the squinting eye, while the patient's attention is first directed to the candle to note which eye is the fixing one, or, in other words, to note on which eye the corneal image of the flame occupies the 'fixation position,' usually a little to the inner side of the centre of the pupil. The amount of squint being guessed approximately, the patient is told to look at that figure which represents the guess, then at one or another, as required, till the corneal reflection on the squinting eye occupies the same position as at first on the fixing one." In this way, the patient reads off on the scale the amount of his squint in metre angles (or degrees, if the scale be an ordinary tangent one).

The ordinary laws of the transference of the squint from the one eye to the other, when the fixing eye is occluded, and the equality of the primary and secondary angles by which a concomitant squint differs from a paralytic squint, are not altogether without exceptions. When, for instance, there is a difference of refraction in the two eyes, the accommodation required when one eye fixes is different from that which is required by the other for the same distance, and consequently the same association between accommodation and convergence leads to a difference in the relative directions of the two axes of vision, according as one or the other fixes. A very common instance of this is the case where one eye is myopic, while the other is emmetropic. The emmetropic eye is used for distant fixation, and there is then, it may be, no squint. For near vision it is frequently, on the other hand, the myopic eye which is used, while the other diverges. If, however, the myopic eye be occluded, and the emmetropic one used for fixation, the *secondary* deviation (behind the occluding hand) is either considerably less or absent altogether. Often there is found to be in such cases divergence both for near and distant vision, the myopic diverging in distant fixation, the emmetropic in near. Sometimes, where the one eye is hypermetropic and the other myopic, convergent strabismus may exist for distant, and divergent for near fixation; and in these cases there is not only not the ordinary transference of squint from one eye to the other, but there is instead convergence when one eye is occluded, and divergence when the other is occluded. We must therefore, in any case, look for the explanation of deviations from the typical conditions in the state of refraction of the two eyes.

A point of some practical importance, as well as of great theoretical interest, is the nature of the amblyopia of the squinting eye. Very different views are held on this point. By some the amblyopia is looked upon as the cause of the squint; by others as the consequence. Those who entertain the latter view consider the defect of vision to result from disuse; hence the name *amblyopia ex anopsia.*

The amblyopia of the squinting eye—that is, the amblyopia in cases where there are no objective appearances to account for it, in which alone there can be any doubt as to the nature of the connection—varies within tolerably wide limits. We may distinguish two main forms:—1. That in which central fixation is retained; 2. That in which it is lost, or in which at any rate there is found to be no supremacy of the central over the peripheral portions of the retina. In the first form we find two elements, one of which is permanent, and the other capable of disappearing when the squinting eye is for some days used for fixation; that is to say, the amblyopia may disappear to a certain variable extent, but rarely entirely. Thus one frequently sees that when, owing to some injury, the good eye has its vision temporarily or permanently impaired, the squinting eye not only takes up the fixation, but rapidly improves in vision, although as a rule falling more or less short of full vision. Moderate degrees of amblyopia of one eye are frequently met with in cases where there is no strabismus.

It is easy to understand how, when the circumstances favouring strabismus exist, the strabismus is much more likely to become manifest if the one eye is amblyopic, and therefore the value of binocular vision lessened. A manifest squint does indeed often suddenly make its appearance when the vision of the one eye is accidently lost or much impaired. There seems every reason, then, to look upon the permanent element in the first form of amblyopia as not only pre-existing, but as one of the chief predisposing causes of the strabismus.

Are we then to look upon the recoverable element as caused by disuse? In cases where the squint is associated with hypermetropia, the hypermetropia is often found to be to a much greater extent manifest in the squinting eye than in the other, when the squinting eye is all of a sudden, on occlusion of the other, forced to fix. Very considerable improvement is got in such cases by the use of the full or nearly full correction. This circumstance, then, in not a few cases, accounts for some part of the defective vision of the squinting eye. But, besides this, and constituting usually the main, or it may be the only, portion of the recoverable element in the amblyopia, is what must be looked upon as a kind of awkwardness in the eye in responding all at once, to the full extent of which it is capable, to the normal impressions. This condition is indeed very similar to right-handedness, in so far as the left hand as a rule, though capable of performing the same feats as the right, is unable from want of practice to do so. The habitual suppression which takes place to a greater or less extent of the images falling on the squinting eye cause it to lose the habit of responding fully to their impressions. Nevertheless a very short practice, when the fixing eye is thrown out of gear, suffices to overcome this. The recognition of the two elements in the amblyopia of the squinting eye, in most cases, at any rate, when the power of central fixation is retained, is in so far of practical importance, that we may thereby see the uselessness of continuing for months, as is sometimes done, to exercise that eye in order to improve its vision.

A true permanent amblyopia from disuse is denied by some, because they say that, for instance, cases in which cataract has existed for thirty or forty years, have not been found to have suffered any loss of vision after a successful operation. There can be little doubt, indeed, that when central fixation has been once acquired it is never lost by circumstances which merely interfere with the formation of distinct images on the macula. It is otherwise, however, with cases in which cataract or any other opacity exists at the time that the supremacy of the fovea over other parts of the retina is properly acquired. When the cause is removed, although a certain amount of vision is restored, there is no central fixation. This is always the case in dense congenital cataract, and often, too, where there has been a long persisting dense corneal opacity following ophthalmia neonatorum. In the first case, after removal of the cataract, and in the second, even when all, or nearly all, the opacity has cleared away, the power of fixation is found to be absent, and always to remain absent. In cases of monolateral strabismus, which begin early in life, that is, during the first months, the squinting eye may be regarded as subjected to the same unfavourable conditions as far as the acquirement of central fixation is concerned. Fixation with that eye is never called for, and therefore never acquired. The second form of amblyopia, viz., that in which there is no central fixation possible in the squinting eye, is in so far an *amblyopia from disuse* in that it is occasioned by disuse at a time when central fixation is usually acquired. Besides the parallel cases of absence of central fixation from early disuse given above, there is one circumstance which argues strongly for the correctness of this view. We find, as already said, very frequently unilateral amblyopia where there is no squint, but rarely if ever an amblyopia without central fixation, *i.e.*, of course in cases where there are no objective signs to explain the absence of central fixation.

When there is, as we have seen is most frequently the case, no diplopia, it is by no means easy to account for why there should be such a total suppression of the image falling on the squinting eye. It is easy to satisfy one's self that that portion of the field of the squinting eye which is not coincident with the field of the other eye is constantly aware of the impressions it receives. The fact seems to be simply that in cases where there has never been binocular vision, and in which usually there is considerable congenital amblyopia, diplopia does not exist, whatever be the explanation. One can hardly draw any conclusions in cases of absence of binocular vision from what occurs when there has been binocular vision. In a number of cases, however, there is another reason for the absence of diplopia, viz., the fact that the squinting eye has acquired the habit of projecting its images in accordance with its position. In these cases there exists sometimes an imperfect form of binocular vision, but more frequently the absence of diplopia is due to the circumstance that, although there is no simultaneous elaboration of the two images of any object, still, whenever the squinting eye is conscious of its image, it is projected to the same place in space as that falling on the fovea of the properly directed eye. That is to say there may be simultaneous vision with both eyes, and yet no binocular fusion of images in the true sense.

The two forms of suppression just referred to, although they hardly account for all the cases met with, are of interest so far as the conditions induced by operative interference are concerned. In the first form there is rarely any diplopia after operations, whereas in the second the operation gives rise to diplopia, of which the patient is more or less conscious. The new position of the eyes is not at first, or for some time, allowed for, and the position being relatively divergent, the persistence of the previous habit of projection results in more or less marked crossed diplopia.

In the *treatment* of convergent strabismus we have to take into consideration the fact that there is a tendency to a gradual disappearance of the squint as the patient grows older. When this does occur it is seldom before the tenth year, and generally considerably later. The nature of the squint, too, whether periodic or constant, relative or absolute, must influence the treatment; so also must the state of vision in the squinting eye, and the presence or absence of diplopia. Usually when some operative measure is required, the degree of the permanent squint will afford an indication as to what operation, or combination of operations, is advisable.

Though much can be done in some cases without operation, the cure of a convergent squint can usually only be thoroughly made by bringing about an alteration in the relative positions of the tendinous attachments of the lateral muscles. The permanent element of the squint can only be cured in this way, whilst that portion which is accommodative often disappears under the altered conditions brought about by proper correction of the existing error of refraction. When the squint has begun late and has not existed long, and where there is either spontaneous or easily elicited diplopia, with not too high a degree of amblyopia of the squinting eye and hypermetropia, an attempt should be made to cure it by causing the patient to wear glasses which as nearly correct his hypermetropia as he will tolerate. By keeping the accommodation paralysed for some weeks during the first wearing of the glasses, a pretty full correction may often afterwards be permanently worn without inconvenience, and a favourable effect on the squint at the same time obtained. Owing to the difficulty of giving spectacles to young children, it is only a small proportion of cases in reality which admit of purely optical treatment.

The continued use of atropine for the purpose of keeping the accommodation paralysed, and thereby avoiding the tendency to squint, which is advocated by some, is seldom of any use, or at all events the benefit in any case is only temporary, lasting as long only as clear vision is interfered with in this way.

The operation of tenotomy of the internus, or advancement of the tendon of the externus, only produces an alteration in the mechanical consequences of the existing relation between the power of the two lateral muscles, but does not influence in any direct manner the cause of the squint. That is to say, that by displacing the attachment of the internal rectus backwards, or that of the external rectus forwards, the initial position of the eyeball is altered in such a manner as to introduce a state of less convergence of the two axes. In this way the appearance is improved, while sometimes at the same time a resumption of function, which the abnormal convergence rendered previously impossible, is permitted. Still the conditions of innervation, which are the cause of the squint, remain. This is evident from the degree of insufficiency of convergence which results from the operation. Indirectly, the operative effect may lead to a true cure of the squint, by diminishing so far the abnormal muscular conditions, that the further difficulties can be more or less readily overcome by fusion. When this happens, there must have of course previously existed binocular vision, and the latent or dynamical deviation always remains. Whether the full permanent amount of the squint, or even more, should be corrected by operation, depends again on the age of the individual, as well as on the existence or not of those circumstances already referred to, which help to maintain the correction when once effected. The object of the operation is not merely to correct the position of equilibrium, but also to do so as far as possible in such a manner as shall admit of the accommodative and associated movements of the two eyes taking place in the normal way.

In operating on children with hypermetropia and amblyopia of one eye, we should leave a little of the convergence uncorrected, owing to the possibilities of a change, in the direction of spontaneous cure, taking place as they grow. One or even two millimetres may be left without, as a rule, being observable, owing to its being more or less covered by the relative divergence of the optic, as compared with the visual axes. In adults we may usually safely correct the whole amount of permanent squint, even where there is no possibility, as is often the case, of their obtaining binocular vision after operation. In cases where the conditions favouring the simultaneous use of the two eyes are more advantageous, and at the same time the degree of convergence associated with accommodation is clearly in excess of the normal, it is advisable to correct the full permanent element. In alternating squints, too, the whole correction should be made.

When the convergent strabismus is associated with myopia, and is only relative, that is, only existing for fixation beyond a certain distance, care has to be taken that the correction of the squint for a distance does not give rise to insufficiency of convergence for near objects. Such insufficiency would either introduce relative divergence with diplopia for near vision, or asthenopia, owing to the too great effort necessary to keep up the required amount of convergence. With correction of the myopia, I believe, this danger need seldom be feared, so long as the position for a distance is not over-corrected. It is well, however, to determine beforehand whether there is any latent convergence at the reading distance. Should there be, there can be absolutely no doubt as to the advisability of performing a tenotomy.

The effect of a single tenotomy in the manner in which it is now performed, *i.e.*, by a mere detaching of the direct sclerotic attachment of the internal rectus, is not always the same, as this depends partly on the nature of the indirect insertions of the tendon, and partly on the degree of rotation which is given to the eye by the antagonist whilst the re-attachment is taking place. The latter depends again on the nature of the squint, on the visual acuity of the eye on which the tenotomy has been performed, and on the use made of the eye during the healing process. It is apt to be less the more amblyopic the eye is. As a rule, however, we cannot expect a greater alteration in the position than about 4 millimetres linear measurement, or a diminution of the convergence to the extent of from 15° to 20°, or 4 to 6 metre angles. In a large proportion of cases in which one has to operate, the position to be corrected is considerably, sometimes greatly, in excess of this amount, and the question arises, how is one sufficiently to increase the effect? Any means which permits of much further retraction of the internus, though it would increase the effect, would at the same time increase the insufficiency of the action of the muscle, and give rise to irregularity in the associated movements of the eyes, and possibly to a relative divergence for near vision. One or two millimetres greater effect might, however, in many cases be desirable, without fear of too great insufficiency. If it were possible with any certainty to regulate the amount of extra effect thus obtained, this could be got by dividing more or less of the indirect fibrous attachments of the muscle, and not only its sclerotic attachment. This plan, which is pretty frequently practised, should as a rule be avoided owing to its uncertainty, the tendency being for the effect to be too great, and the insufficiency consequently too marked.

When a further correction than that which can be obtained by tenotomy of the internal rectus of the squinting eye is required, we have often the choice of two operations—either tenotomy of the internal rectus of the other eye, or advancement of the tendinous insertion of the external rectus of the squinting eye. As a rule the first plan is preferable, as in all but an insignificant number of cases the squint is to be looked upon as an increased convergence of the axes of the two eyes, and not merely a preponderance of the internal rectus of the squinting eye alone over its antagonist. It is, therefore, best to divide the effect over the two eyes. Wherever there is marked weakness of the abduction in the squinting eye and not in the other, a condition sometimes seen when the squint is of paretic origin, the tenotomy should be combined with advancement of its external muscle, as not only is this method of operating calculated to render the relations of the lateral muscles more nearly similar in the two eyes, but under these circumstances the effect of the tenotomy alone on the squinting eye is apt to be smaller than usual. An increase of correction brought about, too, by advancement of the external rectus produces relatively less insufficiency of the internus than a corresponding increase caused by a greater retraction of the internus, so that in this way a gain is effected in the position without too great sacrifice.

The amount of diminution in the power of adduction, resulting from a properly performed tenotomy, must depend on what was previously the position of attachment of the tendon, as well as on the direction of the muscle, that is to say, the point where it becomes a tangent to the eyeball. This defect or insufficiency in the power of adduction is sometimes the same in amount as the correction of the position to which tenotomy gives rise; it is fortunately, however, as a rule rather less, though occasionally it is even more.

In the highest degree of squint, *i.e.*, when the deviation is greater than from 8 to 10 milimetres linear measurement, or from 35° to 45°, or from 10 to 11 metre angles, and the double tenotomy is consequently insufficient to effect a complete or sufficiently approximate correction, the advancement of the external rectus of the squinting eye must be done in addition to the tenotomy on each eye.

A point on which there is a difference of opinion amongst ophthalmic surgeons is as to whether, when it is necessary to divide the effect of operation over both eyes, both should be operated on at once or at different times. Where it is possible, it is better to operate at an interval of at least one week, owing to the uncertainty in the extent of the effect which each tenotomy produces.

When the circumstances are not favourable to an increase of the effect, *i.e.*, when there is not good vision in both eyes or any tendency to binocular fixation, that which is found immediately after operation, or, if the operation be performed under an anæsthetic, as soon as the patient has completely recovered from its effects, is, generally speaking, approximately that which one may expect to see remain permanently. On the day following the operation, and for some days subsequently, the effect is often rather greater, but the gradual tightening of the new adhesions usually eventually brings it back to the first amount. The effect of a tenotomy is immediately, and for some weeks after operation, found to be greatest for fixation in the direction of action of the divided muscle, and least for fixation in the opposite direction. Concomitancy is lost, and only very slowly regained.

When the amount of the squint is a good deal less than 4 millimetres, so that a tenotomy would be likely to have too great an effect, and it is yet desirable to operate, the retraction of the insertion may be, to a greater or less extent, restrained by a conjunctival suture, placed horizontally and involving more or less of the conjunctiva. Such a suture is more often required in the case of the second tenotomy in squints of a moderate amount, though too great to be rectified by tenotomy of the one internal rectus alone. Even should the too great effect be only noticed on the day after operation, the use of the suture will still enable one to remedy it, and indeed, by separating the semi-attached new sclerotic insertion with the strabismus hook, it may be made use of several days after the operation.

In the immense majority of cases the most successful operative interference leads only to a removal of either the whole, or at all events the unsightly portion of the abnormal convergence. The effect, so far as the eyes are concerned, is only cosmetic, and it is rare, comparatively speaking, that any true binocular vision results. Different methods have been devised for the purpose of stimulating the desire for fusion of the two images. Possibly these may in some cases be productive of the desired effect. In most cases they are, however, altogether useless, and it is indeed doubtful whether in the few cases in which binocular vision returns the cure would not have been just as rapid without their aid. The best test for the presence of true binocular vision, or the accurate appreciation of the third dimension, is that which is known as Hering's (see p. 28) There are many cases, however, in which, as already mentioned, the eyes project their images properly in accordance with their position, and which, short of complete binocular vision, acquire the power, sooner or later after operation, of making use of the two eyes together in such a way as to afford a guarantee against the return of the squint, or the subsequent occurrence of relative or absolute divergence. When a prism of say 4° to 6°, with the angle directed inwards or outwards, held in front of either eye, while the other is fixing any object, gives rise to a convergent or divergent movement of that eye, we may conclude that binocular vision of some kind exists.

The *etiology* of convergent strabismus is a point which still presents very considerable difficulties, probably because there are several causes acting singly or conjointly in different cases. The most generally accepted explanation is that which was first given by Donders. According to this view, the necessity for a greater amount of accommodative action for accurate focussing of any object, near or distant, which exists in the case of hypermetropia, when compared with emmetropia, must have one or other of the following consequences. Either the relation existing between accommodation and convergence has to be more or less abnormally dissociated to admit of binocular fixation, or binocular vision has to be given up in order to admit of the degree of convergence being assumed, which is naturally associated with the amount of accommodation which has to be brought into action to allow of accurate focussing. In the first case there may be asthenopia; in the second there is convergent squint. For with the greater accommodation necessary to focus a point at a finite distance from the eyes, there must be, if the normal connection between association and convergence holds good, a greater degree of convergence than is required for the point fixed. This, too, must lead to an inward squint of one eye, as the other must be used for the fixation of the object. Which of these two conditions actually comes about in any case will depend, according to Donders, on the value of binocular vision, so that any circumstance leading to defective vision in one eye would strengthen the tendency which hypermetropia has to induce convergent strabismus. According to Schweigger, on the other hand, the occurrence or non-occurrence of convergent strabismus is dependent mainly on the existence or not of what he calls elastic preponderance of the interni. When such preponderance exists, and it is admittedly more frequent in connection with hypermetropia than with other conditions of refraction, it depends upon the value of binocular vision, as well as on the amount of preponderance, whether or not a manifest squint is the result. The assumed preponderance of the internus over the externus may exist in one or both eyes; in either case the convergence will be transferred from the squinting eye to the other when the squinting eye is used for fixation. This follows because the innervation required to bring the squinting eye from its convergent to its fixing position is necessarily associated with a corresponding innervation of the internus of the occluded, though usually fixing, eye. Where there is anisometropia, however, the amount of squint, when the eye used for fixation is covered and takes up the abnormal position, is often not the same as when the other eye squints, showing clearly that the degree is partly dependent on the amount of accommodation required in each eye for the focussing of the object fixed.

Another explanation which has been offered, and which differs but slightly from Schweigger's, is that the permanent squint is merely the manifestation of the position of equilibrium of the eyes, the position being convergence instead of parallelism. This position of equilibrium, it is held, is assumed when binocular vision from any cause—and the most frequent is of course amblyopia of the one eye—is not sufficiently useful to assert itself against a disadvantage. According to this view, the spontaneous cure of squint takes place when the anatomical position of equilibrium becomes altered in such a way as to cause the externi to become relatively more preponderant.

It seems very doubtful, however, whether the truly anatomical position of rest is ever one of convergence, and more than doubtful if this convergent position is even as marked as it is in strabismus. What has been called by many the convergent position of equilibrium has been found to be most common in hypermetropia, while the divergent position is most frequent in myopia. From the manner in which these positions have been determined, it is evident that they do not correspond to the anatomical positions of rest. Von Graefe long ago pointed out that it is not easy to discover what that position is in any particular case, as the muscles are continually innervated. Apart, too, from this continual innervation, the explanation and importance of which is discussed further on, it is by no means possible to get rid of the innervation which is associated with accommodation. Although with atropine we may paralyse accommodation, we cannot suppress the *impulse* to it, with which some movement of convergence is necessarily associated. That the impulse to accommodation, and not the actual accommodative change, is all that is necessary to give rise to active convergent movements can be easily shown. Thus one may frequently see a squint arise as soon as an attempt has been made by an eye whose ciliary muscle has been paralysed by atropine to see any object distinctly, even although there is at other times no misdirection of the axes. From the direction of the orbits, and from the divergence met with under complete anæsthesia, it certainly appears as if the anatomical position of the eyes were always a more or less divergent one. This position, too, which must be looked upon as the true starting-point for innervation to convergence, no doubt differs, and perhaps not inconsiderably, in different individuals.

Donders' view, though corroborated in the main by many cases, is principally faulty in that it presupposes a connection in the case of hypermetropia between accommodation and convergence, exactly similar to that which exists in emmetropia. That such is not always, nor indeed most frequently the case, has already been explained. Further, the same fact is shown by the cases in which hypermetropia and unilateral amblyopia exist without convergent strabismus. But whilst the connection between accommodation and convergence is so far from being always what might be called normal, there is yet always some sort of connection, most frequently one in which the degree of convergence is considerably less than that of accommodation, but also one in which the reverse is the case. There can be little doubt that it is wrong to suppose, as has been done, that such a connection takes place only where there is binocular vision. Again, Donders' view does not account for the combination of emmetropia and convergent squint, or for the less common but not rare form of myopic convergent squint. The existing association between accommodation and convergence in any given case is no doubt to a great extent acquired, but it is not unlikely that both the lax condition of such association which would counteract, as well as the more intimate connection which would favour squinting, are in some measure the result of inheritance. It may consequently be in this way that the hereditary disposition to squinting comes into play. Schweigger's view, on the other hand, appears to me to ascribe too much to the mere physical state of the muscles, and to too completely disregard the conditions of their innervation.

In many cases the squint can be easily shown to be purely accommodative, and only appears when an effort is made to see distinctly. In some of these cases the squint is associated with diplopia, and the individual usually prefers indistinct vision to double vision, and therefore exerts his accommodation as little as possible. In other cases, again, the conditions are so unfavourable to accommodation that, although there is never any diplopia on the assumption of the excessive convergence which comes on with accommodation, still an accommodative effort is only comparatively rarely made and never sustained.

The most interesting cases of purely accommodative squint are, however, the regularly periodic cases. The squinting position may, for instance, only be assumed every other day, or it may be only in the evening; while at other times the axes are properly directed. The explanation of such cases is evidently that the dissociation of the combined impulses to accommodation and convergence requires an amount of effort which cannot be indefinitely sustained. Rest is obtained therefore by assuming the squinting position for a day.

Frequently, too, one sees cases where the strabismus only manifests itself when the individual is in any way exhausted, but is overcome after food or sleep.

A similar explanation applies to such cases where, after debilitating illnesses, a manifest squint makes its appearance. In some such cases, although there may be good vision in either eye, and diplopia, it is found on testing with prisms that the power of fusion—that is to say, of dissociating the associated impulses to accommodation and convergence in the interest of binocular vision—is very slight. Yet the absence of any squint before the illness shows that the effort required for such a dissociation has previously not been too great to be permanently maintained.

The *permanent* element of the squint, the amount of which, in so far as it alone, as a rule, calls for operative correction, it is of some importance to ascertain, at all events approximately, is at the same time that which is most puzzling in its nature. Several explanations have been advanced. Alfred Graefe, for instance, believes that an actual shortening, due to structural change, takes place in the internus of the squinting eye; while Schweigger holds that there is gradually induced in this muscle an increase in the amount of its tonic contraction, whereby it becomes permanently although only functionally shortened. Neither Alfred Graefe, however, nor any one else, has as yet furnished an anatomical demonstration of the supposed structural change in the muscle. The fact, too, that a permanent squint may altogether disappear in the course of time, while it does not, of course, prove that such a change never takes place, does most decidedly show that it does not always take place. Schweigger's explanation gets over the difficulty of the spontaneous cure of strabismus. Both views are, however, inconsistent with one circumstance in connection with squinting, viz., the disproportion which almost always exists between the deviation of the squinting eye and the insufficiency of its outward movement. Although in the great majority of cases of convergent strabismus some restriction in the extent of possible outward movement is evident either in one or both eyes, the restriction is rarely if ever equal to the amount of the squint, and is generally very markedly less.

From the fact that a permanent squint is only very gradually acquired, and may altogether disappear under an anæsthetic, it seems most natural to assume that it is only the expression of an altered condition of permanent innervation brought about by the disuse of the stimuli, which, as long as the two eyes always work together, succeed in counteracting the tendency to over convergence from association with accommodation, or from any other cause. We have, in fact, to distinguish between an equilibrium of the lateral muscles, independent altogether of the state of their innervation—what has been referred to as the anatomical position—and a position of innervation equilibrium. The requirements of an individual possessing good binocular vision are such as to favour the acquirement of a position of innervation equilibrium, in which the visual axes are parallel, and this altogether independent of what may be the anatomical position of rest. On the other hand, the constant necessity for over-convergence which is seen in hypermetropic individuals who squint leads to a permanent innervation equilibrium of convergence, the degree of which may not unlikely depend to some extent on what is in any particular case the position of anatomical rest. We may say, then, that permanent squinting comes from squinting, just as permanent straight vision comes from looking straight; the state of constant innervation is in fact in accordance with the continued habit in each case.

This explanation, which makes squinting a mere phenomenon of innervation, and not due to any muscular defect, is that given by Hansen Grut. It is certainly more consistent with all the clinical facts in connection with strabismus than those which have been mentioned. As far at any rate as the most frequently occurring cases, where the squint is connected with hypermetropia, is concerned, it is but an extension of Donders' view to its natural conclusion. There are, however, other cases in which the original cause of the abnormal convergence is less apparent, in which, for some reason or other, convergent movements are greatly in excess of accommodative changes. Whatever may be the cause, though, of the squinting, the tendency to its becoming permanent is explained by the tendency that there is for the state of permanent innervation to lead to a convergent state of equilibrium. That the deviation met with in strabismus is an innervational one is evidenced by the extreme cases of insufficiency of convergence, or actual loss of converging power, which are met with after the correction of high degrees of convergent strabismus. In such cases there still exists a power of moving either eye inwards, in association with the outward movement of the other eye, though naturally to a less degree than before operation. With a parallel position of the eye, therefore, there is nevertheless a complete, or nearly complete, innervation to convergence at play.

This *innervation theory*, as it may be called, explains the concomitance of the squint, the equality of the primary and secondary deviations where there are no great differences in the refraction of the two eyes, and the restriction which is met with in the extent of possible outward rotation of the squinting eye. We know, as has been very clearly demonstrated and explained by Hering, that an object in front of or to either side of the eyes, and at the same distance from them, necessitates always the same amount of convergent impulse, although in one case the eye is turned inwards, while in another the same eye is turned outwards. In the first case the inward rotation of the eye is due partly to a contraction of the internus actuated by a convergent impulse, and partly to one brought about by an impulse to associated movement with the opposite externus. In the second case, again, while the contraction of the internus as the result of a convergent impulse is still present, it is more than counterbalanced by the externus contraction, which obeys the associated impulse: that is to say, the eyes follow each other to either side without any alteration in the amount of convergence. In the same way, then, in the case of a convergent squint, as the innervation to convergence, associated with a particular distance accommodated for, remains the same, no difference of any amount in the degree of the deviation is met with to either side—that is, there is concomitance. Further, the external rotation of the eye must be either greater in range or effected with greater ease when unresisted by the contraction of the internus, than when a convergent impulse causes a counteracting contraction of that muscle. We might therefore expect to find some restriction in the outward movement when near objects are fixed, when compared with that possible on the fixation of distant objects. To some extent, no doubt, the restriction found to exist in the outward rotation is merely apparent, and due to a want of effort to bring about a position which is never called for. The idea of a permanent convergent innervation as the cause of squint is certainly, then, supported by the restriction of the external movement, which is often apparent in the non-squinting as well as in the squinting eye.

DIVERGENT STRABISMUS.

Some degree of latent divergence for distant fixation is more frequent, in adults at least, than latent convergence. When from any cause one eye in the case of an adult is rendered blind, it is therefore more frequently found to fail in the direction of insufficient convergence with respect to the object fixed, or, in other words, to diverge, than to take up a relatively too convergent position. The latent divergence becomes in fact a manifest divergence when binocular vision is rendered impossible by the loss or considerable impairment of vision of one eye. The degree of divergence, too, increases as time goes on. This is no doubt owing to the alteration in the state of permanent innervation which the defect in convergent movements brings about. Such convergent movements become defective owing to the necessary absence of fusion. The total extent of the divergence still existing after a long time differs, too, in different cases, and this may not unlikely be owing to the differences in the state of anatomical equilibrium.

The reason why divergent strabismus is so much less common than convergent, is that so many cases of squint arise in early life, after congenital or early acquired amblyopia, or other conditions leading to absence or loss of binocular vision, at a time when the conditions of innervation favour convergence rather than divergence.

Divergent strabismus, absolute as well as relative, and constant as well as periodic, occurs in association with all conditions of refraction; but whilst convergent strabismus is most frequently associated with hypermetropia, divergence is, on the other hand, though not to a corresponding extent, associated with myopia. Where there is myopia the diverging eye is very frequently much more myopic than the other, and amblyopic at the same time. Whereas in the case of hypermetropia there is apt to be a tendency to over-convergence, in myopia the requirements are such that convergence receives little or no support from accommodation; and this circumstance may give rise to what is in reality an insufficiency of convergence, though it is often wrongly spoken of as insufficiency of the internal recti. This insufficiency, along with any cause which diminishes the value of binocular vision, is an element of importance in the etiology of divergent strabismus.

Relative manifest divergence is often seen when one eye is myopic, although not to a high degree, and with good visual acuity, and the other either emmetropic or hypermetropic, or it may be only slightly myopic. The myopic eye is used then for the fixation of near objects, and the other diverges. The different states of refraction render it impossible to obtain binocular vision in reading, and as the reading can be done by the myopic eye without any exertion of accommodation, it is almost invariably used. There is therefore nothing to keep up a proper direction of the other eye, as with the absence of accommodative impulse there is no associated convergent impulse. Just as in hypermetropia the relation between accommodation and convergence tends towards over-convergence, so in myopia it tends towards too great divergence; and as in the first case we have seen that when once the excessive convergence has become manifest it tends to increase, so in the case of divergence the disuse of the counteracting conditions of innervation lead gradually to an increase in its amount when it has passed from the latent to the manifest form.

Two important forms of divergent strabismus should be distinguished: one in which, notwithstanding the divergence, convergent movements take place along with the other eye; the other in which such convergent movements are almost entirely absent, and the diverging eye only moves in association with the lateral movements of the other. In the latter case either the prolonged absence of the usual stimuli to convergence has led to its gradual disappearance altogether, or something has occurred to interfere with the central innervation which should bring about movements of convergence.

The operations which may be required for the cure of divergent strabismus are tenotomy of one or both external recti, with advancement of the internal rectus of the squinting eye. It is rare, indeed, that the internal rectus of the other eye as well requires to be advanced. The conditions in the case of divergent strabismus are somewhat different from those in convergent strabismus: insufficiency of the external recti after tenotomy is, owing to the large extent of the portion of the tendon applied to the globe, less likely to occur, and even where it does, as long as parallelism is possible, it is of less importance than insufficiency of the internal rectus. In most cases it is best, therefore, to attempt to rectify the faulty position by at once performing tenotomy of the external and advancement of the internal rectus in the squinting eye. This, too, is all the more advisable, as in many cases the effect to be got by the tenotomy alone is slight compared with that which usually results from tenotomy of the internus.

When there is good vision in both eyes, tenotomy of one or both externi is often sufficient to secure a good correction. The most unsatisfactory cases to operate on are those in which the convergent movement has been lost, as in them, even although the position be corrected for distant fixation, the relative divergence when nearer objects are fixed always remains. What has been said with reference to the ultimate result of operations for convergent, applies in the main in the case of those for divergent strabismus as well.

Upward or downward squints are, as already said, rarely seen alone, though not infrequently met with as a complication of lateral deviations. They often remain after full operative correction for the convergent strabismus, though they sometimes disappear with it. It is not improbable that their pre-existence may give rise sometimes to the lateral forms, where the other conditions favour convergence or divergence. It is rare that any operation is called for in the case of vertical squints, although tenotomy of either superior or inferior rectus may sometimes be performed.

Operations of this kind have been performed of recent years, apparently very frequently by some American ophthalmic surgeons. The reason why such interference, which is unquestionably altogether uncalled for, does not lead, as a rule, to any serious consequences, is that most vertical deviations are of the same passive nature as latent divergence, so that no great retraction of the divided muscular insertion takes place. The beneficial results following occasionally, at all events temporarily, tenotomies of the superior and inferior recti, are, no doubt, due mainly to rest.

In a few cases of vertical squint, the deviation is of another nature, more allied to convergent strabismus, in so far as the muscles are in a state of contraction. The proper regulation of operations undertaken for the relief of such conditions is very difficult. They probably result from some vicarious contraction induced, in the first place, at a time when there has been a paretic condition of one of the lateral muscles. But their pathology, in all cases, is far from clear.

NYSTAGMUS.

Nystagmus is the name given to involuntary oscillatory movements of the eye. The movements are mostly from side to side, but may be rotatory or almost entirely vertical. They are generally constant, but increase in intensity with attempts at fixation, or when the individual is in any way excited. In some cases the nystagmus only exists for certain directions of fixation, most frequently when the eyes are directed upwards. There is always some degree of jerky or nystagmic movement whenever the eyes are forcibly turned in any direction, and an attempt made to maintain them for any length of time in what corresponds to the boundaries of the physiological action of the respective muscles. Nystagmus is consequently a frequent symptom in ocular paralyses, when the eye is moved in the direction of the weakened muscle.

Most cases of nystagmus are developed in early life as the result of defective vision at a time when the movements of the eyes suited to the requirements of fixation would otherwise be acquired. The education of the co-ordinating centres is thus imperfect. Sometimes these centres appear to be primarily affected, but little is known as to the cause of this. Nystagmus may be acquired in after life as the result of changes in the nerve centres, or as a consequence, it would seem, of some altered states of innervation brought about by abnormal conditions of illumination and fixation.

A common variety of acquired nystagmus is what is called *miners' nystagmus.* In this condition the irregular involuntary contractions take place mainly or entirely in looking upwards, and are associated with the subjective symptom of a corresponding rapid movement of external objects which is very disturbing. There can be little doubt that miners' nystagmus is caused by the tiring of the ocular muscles, which are overstrained owing to the constrained position in which the eyes have to be maintained by the miner at his work. Snell has shown very conclusively that it is only developed in those whose work is of such a nature as to necessitate the strained position of fixation. When this constant straining is given up the nystagmus slowly passes off. In this respect the miners' nystagmus differs from that which is acquired owing to defective vision in early life. In the latter, too, there is seldom any subjective sensation of movement of external objects.

SECTION III.

CHAPTER XVIII.

OPERATIONS.

General Remarks.

Operations are performed on the eye, or its appendages, with various objects in view. The most important, and, when they prove successful, perhaps also the most satisfactory operations, are those which aim at the improvement of the state of vision, or the prevention of any threatened deterioration of vision. Some operations are performed solely or mainly for the relief of pain; others again seek to remove diseased conditions which endanger life, or to remedy deformities which entail danger to the eye, while some are mainly intended to have a cosmetic effect.

Before proceeding to a detailed description of the various most suitable operations, a few general remarks as to the precautions which should be taken in all cases may not be out of place, and will at the same time serve to obviate too much repetition.

All the instruments used in eye operations must be absolutely clean. They must not only be aseptic, but free from rust, blood stains, or any foreign matter. This applies most urgently to such as are, for any purpose, introduced into the eye. Any little piece of foreign matter, even though aseptic, if it becomes lodged within the eye, is capable of seriously impairing the success of an operation. Little bits of cotton or linen fibre may easily escape detection, and for this reason the instruments should not be rubbed with lint, cotton wool, or any material from which fine fibres might become detached. Instruments may be rendered aseptic in various ways, and it may not always be possible to adopt the same method. Some antiseptics, though sufficiently strong, spoil the steel, others are irritating to the eye. Boiling water, or a five per cent. solution of carbolic acid are the most practically useful; but before using the instruments after they have been rendered aseptic by carbolic acid, they must be transferred to some non-irritating solution. For this purpose boracic acid, in the strength of 1 to 50, may be used.

Besides being clean and aseptic, it is of considerable importance to see that the cutting instruments, knives, scissors, needles, &c., are sharp. A sharp instrument is a desideratum in most surgical operations, but it is very essential often to the success of an operation performed on the eye, as not only is the manipulation of a sharp instrument much easier, but the greater dragging, bruising, and stretching, which may be caused by blunt steel, is not favourable to healing by first intention, and may give rise to troublesome irritation. At the same time as the vitality of the tissues is interfered with to a greater extent than in the case of clean cut wounds the danger of septic inoculation from any existing cause is increased. The points and edges of the knives should be tested immediately before they are used. This is best done with a little piece of thin kid leather kept tightly stretched by an assistant, or stretched across a small drum made for the purpose. To a sufficiently sharp point this should offer no appreciable resistance, and the keenness of the edge may be inferred from the ease with which the leather is cut by it. Scissors, besides being sharp, should have their blades sufficiently tightly screwed together to cut readily at the points.

It is necessary to have a good light on the eye whilst operating. The table or couch must therefore be so placed that the operator or his assistants do not intercept the light, which should preferably come from one side alone. When artificial light is required, the rays from a paraffin lamp may be focussed on the eye by means of a large convex lens held by an assistant. A useful little lamp for this purpose has been invented by Priestley Smith. It is practically a small bull's-eye lantern, which affords an excellent illumination, and is easily held in a convenient position for operating. Incandescent electric lanterns have been devised, and are in use, but do not offer any advantage over Priestley Smith's lamp, and are besides much more expensive. During an operation on the eye itself, care must be taken that the patient's head is firmly held. This may be done by an assistant, who should kneel in order to be out of the way of the operator. With one hand on either side of the head it may be securely fixed. Another plan, often adopted, is for an assistant on either side to hold one of the patient's ears, or the two ends of a cloth passed over his forehead. Some operating chairs and couches are fitted with an arrangement for fixing the head.

The introduction of *cocaïne* as a local anæsthetic has in great measure done away with the employment of general anæsthesia in eye operations. The more important operations for the improvement of vision can now generally be performed with the aid of cocaine, without causing any pain; so that the question as to whether general anæsthesia was advisable or not in such operations, a point on which different opinions were held, no longer calls for discussion. A solution of muriate of cocaine, of a strength varying from 2 per cent. to 10 per cent., is used to drop into the conjunctival sac. The solution should be made up with some antiseptic (corrosive sublimate or boracic acid), so as to obviate the possibility of introducing any micro-organisms into the eye. When the stronger solutions are used the point of the finger should be pressed against the skin over the tear sac to prevent the cocaine passing down into the nose, and from there to the stomach. The patient should be told to keep his eye closed after the drops have been applied. If this is not done, there is a tendency to a rapid dessication of the surface layers of the cornea, on account of the insensibility which the anæsthetic produces checking the normal blinking, and probably also the flow of the natural lubricants.

The value of *antiseptics* in eye operations is now so definitely established that it is nothing short of inexcusable not to make use of them. Septic inoculations cannot invariably be avoided by such antiseptic precautions as it is possible to take, although their frequency can be greatly diminished. The reason of this is, in all probability, that the worst sources of danger are not external to the eye, but in the eye itself. The use of antiseptic applications in the manner about to be described, and the attention to absolute cleanliness and asepticity in the instruments already insisted upon, is no doubt sufficient to prevent the possibility of infection from without. It is otherwise, however, with any micro-organisms which may be lodged in the conjunctival sac, or which may find their way into it from the tear passages.

They cannot be destroyed with certainty, and the tying up of the eye even with an antiseptic bandage not only does not exclude them, but rather tends, owing to the retention of the secretions, to favour their development or multiplication. In cases, therefore, where their existence is more than usually obvious, as where there is a chronic conjunctivitis or blenorrhœa of the sac, it is necessary to adopt special antiseptic precautions, and sometimes, if possible, to defer operating until there is reason to believe that such local sources of danger have been removed or mitigated. The most useful antiseptics for local application are solutions of corrosive sublimate, and iodoform. The former is mostly used in the strength of 1 to 5000. In this dilution it is almost always found to be non-irritating, though cases do occasionally occur where some irritation is caused even by such a weak solution. It has been shown by Sattler that a solution of that strength is a sufficiently powerful germicide, and clinical experience fully bears this out. Stronger solutions may be used where there are unusual risks. According to Sattler, a solution 1 to 10,000 of the biniodide of mercury in one of 1 to 5000 of the bichloride is still more powerfully antiseptic. I have frequently used such a solution freely, and found it to be non-irritating and apparently at least just as efficacious as the other. Solutions of the biniodide of mercury alone are not so good, although they are recommended by some. The watery solutions are too weak to be sufficiently powerfully antiseptic, and stronger solutions in other solvents are more often, in my experience, irritating. According to bacteriologists, iodoform is a very poor antiseptic. No doubt this is the case, but it has the advantage of remaining longer *in situ*, and of, to some extent, acting as a cement between the lips of a wound, properties which, partly at all events, make up for its feebler antiseptic properties. A stronger non-irritating antiseptic of the same kind is a desideratum which has not yet been discovered.

Before performing any operation, the conjunctival sac should be thoroughly washed out with corrosive sublimate solution, and the surrounding skin, eyelashes, and eyebrows also carefully cleansed. From time to time, too, during an operation, the lotion may be brought in contact with the eye either by pouring it in a small stream from a narrow-necked bottle, or by squeezing it from a piece of absorbent cotton wool. The antiseptic lotion probably acts in two ways—first as a germicide, and secondly by washing away from the wound the fluid which contains the germs. It is not unlikely that the latter is its most efficient, perhaps indeed its only efficient, action. The methods of dressing will be referred to in connection with the separate operations. Beyond the precautions referred to, viz., attention to asepticity of instruments and of anything brought in contact with the eye, and the use of frequent irrigation, nothing further is required in the way of antiseptics. Some few operators as, *e.g.* Hirschberg of Berlin, attach importance to operating in an aseptic or supposed aseptic atmosphere. To do this they take the most elaborate precautions in the arrangement of their operating rooms, use a special dress for operating, &c. Such precautions are simply an evidence of an inability to understand the true nature of the sources from which the risks of septic inoculation arise. They do not lead to any further reduction in the number of inoculations than a conscientious attention to the simple precautions which are recommended against the real sources of danger, viz., the introduction of septic matter by the hands, lotions, or instruments, and the self-inoculation which may take place from the secretions of the conjunctiva and tear-sac.

OPERATIONS ON THE EYELIDS.

Repositio ciliorum is a very old and elegant method of rectifying the position of faultily directed eyelashes. It may be employed when only a few are misdirected. A small curved needle, through the eye of which the two ends of a fine silk or horse-hair suture are threaded, is passed in through the margin of the lid close to and in front of the root of the misdirected eyelash. It is then brought out through the skin in the immediate neighbourhood of the lid margin. The thread is pulled through until only a small loop remains. This loop is then made to surround the lash, which with it is pulled through the channel made by the needle, and thus altered in its direction. Care must be taken not to pull on the eyelash too forcibly while getting the loop round it, so as not to tear it out by the root. This method does not always lead to the desired effect of producing a permanent improvement in the direction of the eyelash;

indeed the effect is often only temporary. A more efficient little operation is the following.

Destruction of the hair follicles with the actual cautery.—Fig. 175 represents the form of the cautery generally used for this purpose. Electrolysis has also been employed in the same manner as for the removal of superfluous hairs elsewhere.

Epilation with a pair of cilia forceps is often all that is required, as it can be done by the patient's friends or even by the patient himself, whenever the lashes begin to grow. Not infrequently repeated epilation brings about the cessation of the growth of the lash altogether.

Operations for Entropion.

In cases of spasmodic or senile entropion of the lower lid, where a sufficient improvement is not obtained by means of collodion or plasters, the inversion of the lid margin may generally be corrected either by a thread operation or by the removal of an elliptical piece of skin. When these are not sufficient, as is sometimes, though only rarely, the case, it is necessary to perform a canthoplastic operation in addition.

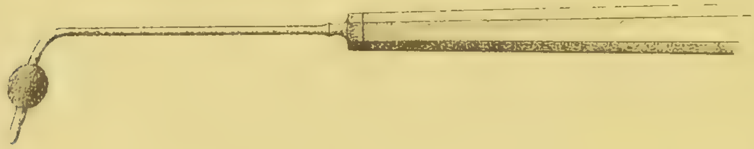

Fig. 175.

Thread operation for spasmodic entropion (*Gaillard's*).—A fold of skin is caught up with a pair of T-shaped forceps, or simply between the forefinger and thumb. The size of the fold should be regulated by the effect which it is desired to produce, but should always be big enough to give rise at the time to a distinct eversion of the lid margin. A needle threaded with a piece of silk or catgut is then passed from below upwards, through the base of the fold of skin, and at about the junction of the middle with the inner or outer third of the lid. The needle is brought out through the skin close to the lid margin and then entered again, at a distance of one-eighth of an inch or so from its point of exit, and carried back below the flap, and finally brought out about quarter of an inch from where it was first introduced. A second thread is then introduced in exactly the same way, at about the other end of the middle third of the lid aperture. The two loops thus formed are tightened by knotting the ends of the threads together over pieces of drainage tubing. If silk threads be used they may generally be removed after two days. Catgut ones, on the other hand, may be left until they fall out. In my experience the former are preferable.

Another thread operation, the effect of which is less permanent, is performed as follows:—A small portion of skin near the lid margin is included in a suture, one end of which is then cut short. Another small bit of skin, lower down over the orbital margin, is caught up with a thread in the same way, the one end of which, after being knotted, is cut short. By then knotting together the two ends which have been left long, the two points of skin are approximated, with the result that a traction is brought to bear on the inverted margin of the lid. Two sutures applied in this manner are usually required. This little operation is very favourably spoken of by Arlt, but as I have always found the one first described perfectly efficient, I have no personal experience of it.

Excision of a fold of skin.—When the skin is very markedly redundant, the simplest method of rectifying the entropion is to remove an elliptical piece. The amount to be removed may be calculated from the effect produced on nipping up a fold in the manner already described. Three sutures are afterwards employed to bring the edges of the wound together, and permit of healing by first intention. The only thing to take care of is not to produce too great an effect to the inner side, so as not to get a permanent eversion of the punctum lachrymale. Sometimes, instead of a horizontal fold, one or more elliptical portions of skin are removed in a vertical direction. This is rarely as satisfactory, and more likely to leave visible cicatrices.

Operations for Trichiasis and Cicatricial Entropion.—The performance of these operations is greatly facilitated by the aid of what is known as Snellen's clamp, an instrument by which the circulation in the lid is stopped, so that the operator can see exactly what he is doing.—(See Fig. 176.)

The misdirected eyelashes may be removed altogether, without shortening of the lid, by *Flarer's operation.* By an intermarginal incision all the tissues in front of the tarsus are dissected up as far as the ends of the hair bulbs of the cilia. The lid is thus split into two portions for some distance from its margin. The anterior flap formed in this way is then cut away by an incision carried through the skin from above. When some of the misdirected eyelashes are close to the outer canthus, it is necessary to extend the marginal incision beyond the canthus by carrying it horizontally outwards to the extent of a quarter of an inch or more, while the upper incision is at the same time enlarged so as to meet it. Care must, of course, be taken that all the hair bulbs are completely removed, otherwise some of the cilia will afterwards grow in again.

This operation, which was at one time very much practised, while it effectually obviates the irritation produced by the turned-in eyelashes, is now almost entirely abandoned, on account of the unnecessary mutilation which it causes. It is generally possible to transplant the portion of the lid containing the hair bulbs in such a manner as to produce a permanent improvement in their direction. Flarer's operation is only suitable in cases where most of the eyelashes are already lost, and those which are retained are bristly and discoloured. It may also be combined with other methods of operation in cases of distichiasis, the inner row being removed while the outer is transplanted.

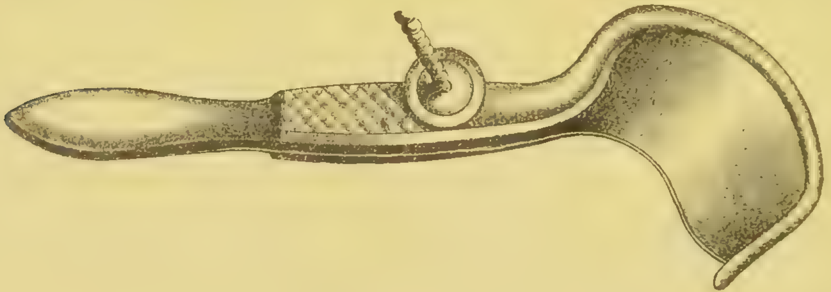

FIG. 176.

Those who previously attempted the cure of trichiasis by removal of the tissues containing the hair bulbs did so by cutting away the whole thickness of the lid. This not only produced an unsightly shortening of the lid, but apparently often gave rise to irritation owing to the inversion of the cicatrix formed.

Von Graefe's Operation.—A method of operating for trichiasis, which is both simple and efficient, and which was first recommended by von Graefe as a modification of Arlt's operation, is the following:—After splitting up the lid from its margin into two flaps, as in Flarer's operation, two incisions, vertical or slightly diverging, are made from either extremity of the flap containing the hair bulbs, and which is to be transplanted.

These incisions should extend for one-third of an inch upwards through the skin and orbicularis muscle. The outer and inner edges of the skin flap are then stitched to about the middle of the vertical incisions on either side, and the edges thus considerably raised. The rest of the flap is raised by the excision of an elliptical portion of skin from the lid, the edges of the wound caused by which are brought together by stitches. This operation is also very suitable for cases of partial trichiasis. In bad cases of distichiasis, von Graefe's operation may be combined with canthoplasty.

The idea of transplanting the cilia in this manner seems first to have occurred to Jaesche, a Russian, in 1844. Jaesche's operation consisted in separating the lid margin altogether, except at its outer and inner ends, from the rest of the lid by means of an incision slanting upwards from the conjunctival to the skin surface, and then raising the quadrangular portion thus separated, and with it the hair bulbs, by removing a good large piece of skin from the surface of the lid, and bringing the edges of the wound made in this manner together with sutures. This operation was found to be pretty often followed by necrosis of the transplanted lid margin, and was therefore modified by Arlt, who, instead of entirely separating the marginal portion of the lid from its conjunctival to its skin surface, substituted Flarer's incision, dividing it into an anterior and posterior flap. The operation performed in that way generally goes by the name of the *Jaesche-Arlt operation for trichiasis*, and is still in use. It has, however, the disadvantage that while it sufficiently rectifies the position of the lashes at the centre of the lid, it effects less change on those situated near the angles.

Another method of operating for trichiasis, which gives very good results and is easy of execution, consists in uniting the skin above the lid margin to the upper border of the tarsal cartilage. This, which seems first to have been practised by Anagnostakis, has recently been revived by Hotz. *Hotz's operation* is performed as follows:—A cut is made through the skin of the lid on a level with the upper border of the tarsus of the upper lid, or the lower border of the tarsus of the lower lid.

A layer, 3 to 4 millimetres in breadth, of muscular fibres overlying the tarsus is then excised. The edges of the wound in the skin are finally brought together by stitches, which are passed through the upper border of the tarsus. The two points to be attended to in order to ensure success by this operation are—(1) the position of the section in the skin, which must throughout be on a level with the upper border of the cartilage, and (2) the proper position of the sutures, which must firmly include the upper edge of the tarsus. Hotz claims the following advantages for this method:—that it attains the aim in view without the least shortening of the skin, and on this account may be employed in cases in which, owing to the shortening produced by previous operations, other methods are not available; that it in no way interferes with the shape or function of the lid; and, finally, that the stretching of the skin which restores the margin of the lid to its proper position is the same whether the lid is raised or depressed, which is not the case when a piece of skin is excised.

Many other operations for trichiasis are in use, but either of the methods above described, if properly performed, serves the purpose admirably. Personally, I am in the habit of performing von Graefe's when there is plenty of skin, and Hotz's, or some modification of it, in cases where it is undesirable to remove any skin. The latter cases are generally such in which some skin has previously been removed, but in which, owing to inattention to the other details, a sufficiently permanent effect has not resulted.

In cases of entropion, when there is much curving of the tarsus, it is generally advisable to perform a different operation, the object of which is to rectify as much as possible the cicatricial incurving. The operations best suited for this purpose are those of Streatfield and Snellen.

Streatfield's operation, to which he gave the name of "Grooving of the fibro-cartilage," is performed as follows:—An incision, parallel with the margin of the upper lid, and 2 or 3 millimetres from it, is made through the skin. A second incision is then made higher up, which throughout the greater part of its extent is also from 2 to 3 millimetres separated from the first, but joins it at either extremity. The portion of skin thus marked out is next removed along with the muscular fibres underlying it, and the tarsus laid bare. The incisions are then extended in depth into the tarsus, being at the same time converged towards each other so as to meet at or near its lower surface. The complete removal of the wedge-shaped portion of tarsus marked out in this way is then effected by grasping it at one end with a pair of fine-toothed forceps, and dividing any part where the incisions have not quite met. No sutures are used, the wound being allowed to cicatrize. From this proceeding a firm deep cicatrix results, which not only leads to a straightening of the incurved cartilage, but to a tacking down practically of the lid margin in a position in which the eyelashes are more normally directed, and thus a permanent improvement is obtained.

Snellen's operation.—Snellen has improved considerably on the original grooving operation, and his method has come into very general use, and certainly leads to most satisfactory results. After having applied the entropion forceps or clamp, an incision is made through the skin and muscular fibres overlying the middle or most convex portions of the tarsus, and parallel with the lid border. A sufficient portion of the tarsus is then laid bare by undermining the edges, more especially the upper edge, of this incision. A wedge-shaped portion of tarsus is next removed as in Streatfield's operation. After this has been done sutures are inserted in the following manner:—Three threads with a needle at either end may be used. One needle is passed deeply through the centre of the upper lip of the wound in the tarsus, then carried down between the fibres of the orbicularis and the lower portion of the surface of the tarsus, and brought out through the skin above to the margin of the lid. The other needle is passed in the same way under the skin and muscle a short distance from the first, and brought out at the same distance from the lid margin, and about a quarter of an inch to the side of the first. The other two sutures are applied in the same manner to the outer and inner side of the central one and the ends of each of the three loops knotted over pieces of thin drainage tubing, or, as Snellen recommends, glass beads. After knotting, the ends are not cut short, but turned up and secured by means of adhesive plaster to the forehead. No sutures are put in the skin. By means of this operation the rectification of the position of the eyelashes is at once obtained by a straightening of the tarsus. The only point which specially requires attention is to see that the grooving is carried out throughout the whole extent of the tarsus and not forgotten at the two extremities. An operation which is essentially a combination of grooving of the tarsus and Hotz's operation is largely practised in Russia, where cases of entropion are of great frequency.

Another method of operating for entropion may be mentioned. It consists in transplanting mucous membrane into the wound made by an incision at the lid margin. This plan was introduced by van Millingen, and has lately been modified and improved by Benson (*vide British Medical Journal*, February 7th, 1891), who has employed it in a very large number of cases with excellent results. Benson dissects with forceps and scissors a flap 3 or 4 millimetres wide from the mucous membrane of the lower lip, and after carefully freeing it of submucous tissue applies it to the wound in the lid made by an intramarginal incision. The flap is secured in position by means of six or seven sutures. The whole operation is performed without removing Snellen's clamp.

My own experience of this method of operating is limited, but so far as it goes is quite sufficient to satisfy me of its efficacy. My colleague, Dr. Argyll Robertson, has used it more frequently, and obtained excellent results. The only drawbacks which may be mentioned in connection with it, and they are unimportant, are, that it is tedious and that the thickening of the lid margin which it sometimes causes is rather unsightly. Instead of transplanting mucous membrane, some operators have employed Thiersch's method of skin-grafting with equal success.

Canthoplasty is an operation which is useful in cases of chronic inflammation of the conjunctiva and cornea, and which may often advantageously be combined with operations for the cure of trichiasis and entropion. When it is desired for any purpose to effect a permanent enlargement of the lid aperture, it is of course necessary to prevent the opening made in the skin of the lid from healing together again. This can generally be effected by a simple transposition of the conjunctiva in the following manner:—One blade of a strong pair of straight strabismus scissors is passed into the conjunctival sac at the outer canthus, and made to push the tissues in front of it as far as possible. A firm snip is then made with the scissors, the other blade of which cuts through the skin, so that a straight wound is made in the direction of prolongation of the lid aperture. The wound thus made at once gapes, and after the bleeding has been to some extent stopped, three sutures are applied.—(See Fig. 177.) The first unites the centre of the wound in the conjunctiva to the apex of the skin wound, while the other two bring the sides of the conjunctiva thus dragged outwards into apposition respectively with the upper and lower lips of the external wound. In this manner healing takes place between the conjunctiva and skin, and prevents reunion of the divided tissues. No dressing is required.

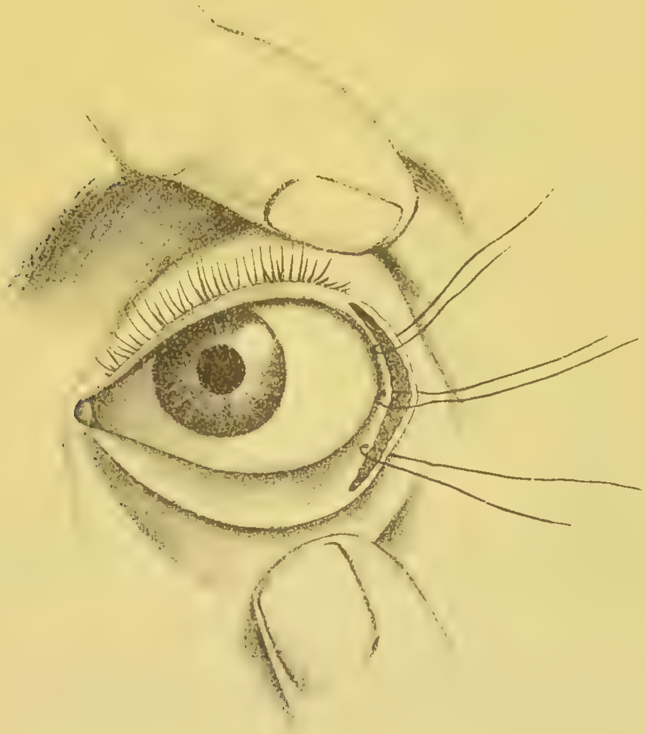

FIG. 177.—(After Wecker.)

This proceeding is rendered possible by the looseness of the conjunctiva which usually exists. Where a similar operation is indicated in cases where the conjunctiva has undergone degenerative changes, and can therefore not be transplanted in this manner, the aperture may be kept open by transplanting skin. An operation of this kind, which gives very good results, was first recommended by Kuhnt. A flap of skin, the shape of which is shown in Fig. 178, is cut from the upper or lower lid. The base of this flap, one quarter of an inch or so in breadth, should have its centre in a line with the lid aperture. The flap should contain nothing but skin. After it has been cut the enlargement of the aperture may be made with the scissors in the manner just described. The conjunctiva is then undermined for a short distance. After this has been done the skin flap is turned into the wound, and its end inserted below the conjunctiva, while the edges of the skin, from which the flap has been cut, are brought together by sutures. An antiseptic dressing is then applied, and left unchanged for two or three days. It is best not to put any stitches into the skin flap, but merely push it under the conjunctiva for some distance, to leave room for shrinkage. It gradually assumes more and more of the character of mucous membrane.

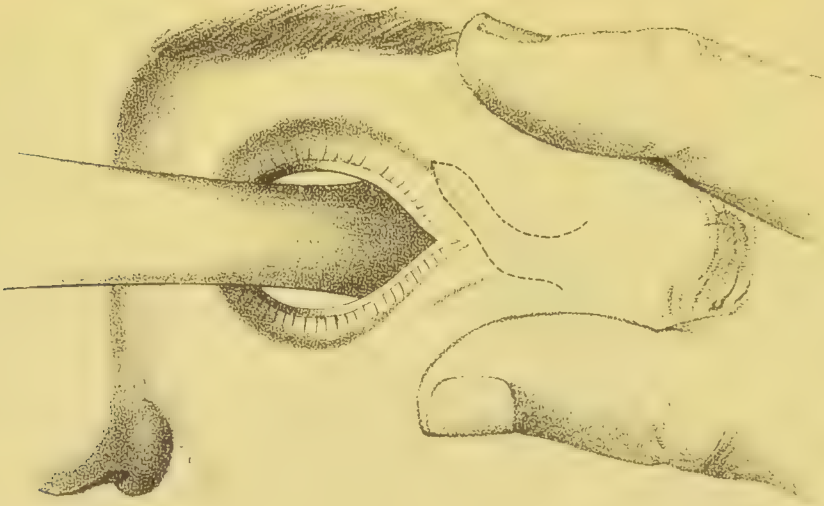

FIG. 178.—(After Kuhnt.)

OPERATIONS FOR CONJUNCTIVAL ECTROPION.

For conjunctival ectropion, or that form which is the result of chronic conjunctivitis and consequent paralysis of the palpebral portion of the orbicularis muscle, two methods of operating may be recommended. These operations are Snellen's thread operation and the excision of a triangular piece of skin from the outer part of the lid. By these methods, either singly or combined, a satisfactory result can always be obtained.

The object of *Snellen's operation* is to exert a traction on the everted conjunctiva, so that it becomes permanently inverted, and thus comes to occupy a more normal position. This he effects in the following manner:—Two strong waxed silk threads with a needle at either end are required. The needles should be curved slightly towards their points, but straight for the greater part of their length, which should be preferably about one inch and a half. One needle is passed through the conjunctiva at a point rather to the inner side of the middle of its inner half. The point selected must be *at the highest part of the everted conjunctiva.* After perforating the conjunctiva it is directed forwards so as to pass towards the skin immediately below the border of the lid. When it has reached this, as can be felt with the finger of the other hand, it is passed downwards below the skin, and brought out as far down as possible, an inch or so below the lid margin. The second needle of the first thread is entered in the same way a few millimetres to the outer side of the first, and brought out at a point a quarter of an inch or so from its point of exit. The second thread is introduced in exactly the same way at the outer half of the everted lid. Two loops are thus formed, the ends of which are firmly tied together over pieces of drainage tubing. The knotting of the threads should be made with sufficient firmness to produce at the time a considerable degree of entropion. According to the effect required, these threads are allowed to remain from two to four days before being removed. In very bad cases three threads may be used instead of two, or the operation may be combined with the excision of a triangular piece from the outer side of the lid.

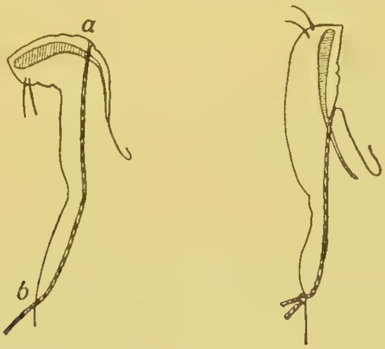

FIG. 179.—Showing position of threads in Snellen's operation before and after knotting.—(After Fuchs.)

The excision of a piece of lid in this situation is in many cases sufficient to correct the ectropion without anything further, and is the best operation to perform in cases where there is a distinct redundancy of the skin of the lower lid. It is not necessary to pare the edges of the lids, but merely to remove a portion of the whole thickness of the lid, and then bring the edges together with several sutures.

Two modifications of Snellen's operation are practised, introduced respectively by De Wecker and Argyll Robertson. Of the first I have no experience; the second can certainly be relied upon to produce the desired effect, but is more complicated and less rapid in its action than Snellen's, owing to the threads being passed through the conjunctiva at the bottom of the fold, between its palpebral and ocular portions, and not at the highest point. It is besides, considerably more painful for the patient.

OPERATIONS FOR CICATRICIAL ECTROPION.

Much will depend on the position and extent of the cicatricial tissue in determining what operation will be most suitable in any given case. When the ectropion is due to a loss of substance which involves the lid above, or when some portion of the skin is tacked down and incorporated with a deep cicatrix at the margin of the orbit, it will generally be possible to rectify it by undermining the surrounding skin and gliding it over the underlying tissue in such a way as to relieve the dragging which causes the eversion of the conjunctiva. The most suitable of the many plastic operations for this purpose is Wharton Jones'. In some cases, again, where the loss of substance has been so extensive that no satisfactory flap is to be got anywhere near the eye, a transplantation from some other part of the body is called for. The success which almost constantly attends this method of operating causes it to take the place to a great extent of plastic operations, which are consequently rarely necessary.

Wharton Jones' operation consists in making a V-shaped incision, with the apex directed away from the margin of the lid, and after freeing the skin thus marked out from the underlying tissues, as well as undermining the skin to either side, putting in sutures so as to unite the wound in the shape of a Y. The size of the V as well as the extent to which the skin is to be loosened will depend on the degree of ectropion. This operation is also usually applicable to cases where the skin is tacked down to the bone, owing to a previous caries; the two incisions forming the V should then embrace the cicatrix. It sometimes happens, however, that it is impossible to dissect up a flap, as the cicatricial tissues are so thinned where they are adherent to the bone. It is then, as a rule, better to cut out this portion altogether, and make up the deficiency which thus arises either by a more extensive transposition of the surrounding skin, or by means of a flap taken from an adjacent part.

When the cicatrix is situated to the outer side of the lower lid, which is its most frequent site, the best operation is probably that devised by *Richet.* This may be performed in the following way:—A curved incision is made through the tissues immediately above the cicatrix, beginning in a line with the palpebral aperture, and as far out as possible, and passing downwards and inwards parallel with the border of the orbit; this incision frees the lid, and permits of the reposition of the everted conjunctiva. Before proceeding any further the upper and lower lids should be brought together by means of three horse-hair sutures; the needles threaded with horse-hair are passed through the upper lid, just at its margin, then through the margin of the lower lid, back through the lower lid margin, close to the first puncture, and finally again through the margin of the upper lid, also close to where the needle was first entered. In this way three loops are formed, which may then be drawn pretty tight together, and which keep the lids in contact. It is unnecessary to pare the margin of the lids, as is often done. After the lids have been brought together in this way, and some gaping of the incision left, another incision is carried from below the cicatrix to meet the first at a point; the cicatrix enclosed is then removed. To fill up the gap thus formed, and which is lettered *C* in Fig. 180, a flap, *A*, is cut, the incisions for which are begun well above the level of the lid aperture. By prolonging the inner incision for the *A* flap, a second flap, *B*, is marked out, which serves to fill the gap left when *A* is twisted into the position *C*.

A moderate degree of ectropion, involving the outer portion of either upper or lower lid, is best treated by the excision of a V-shaped portion from the whole thickness of the lids. The edges of the wound thus formed are then brought together with stitches. The stitches should be close together and deep. Cicatricial ectropion of the upper lid can often be remedied by means of a flap taken from the forehead.

A transplantation operation for ectropion, or the grafting of a large piece of skin taken from some other part of the body, is in many cases the most suitable. When proper precautions are taken the graft always takes. Subsequent contraction of the transplanted skin may, however, to some extent interfere with the perfection of the ultimate result, so that not infrequently in bad cases a second operation of a similar nature is required. Before proceeding to remove the piece of skin to be grafted, which may be taken from any part of the body—most conveniently as a rule from the upper arm—an incision should be made immediately below the border of the upper lid, and a flap formed, containing conjunctiva, tarsus, and muscle, of such an extent as to permit of the ectropion being readily reduced without the least dragging at any point. The upper and lower lids are then brought together, without paring their margins, by loop sutures of horse-hair. A pad of cotton wool, which has been dipped in corrosive sublimate solution, is then placed on the wound, and kept in position by a light bandage until the skin graft is ready. In cutting the graft it is better, I think, not to take too much trouble by attempting to remove it without fat, as the fat can very easily be removed, with greater rapidity and less damage to the vitality of the flap, after it has been cut. The portion of skin taken should, besides being deprived of all subjacent fat, be at least half as large again as the wound into which it is to be grafted. When the wound is very large, two elliptical pieces may be

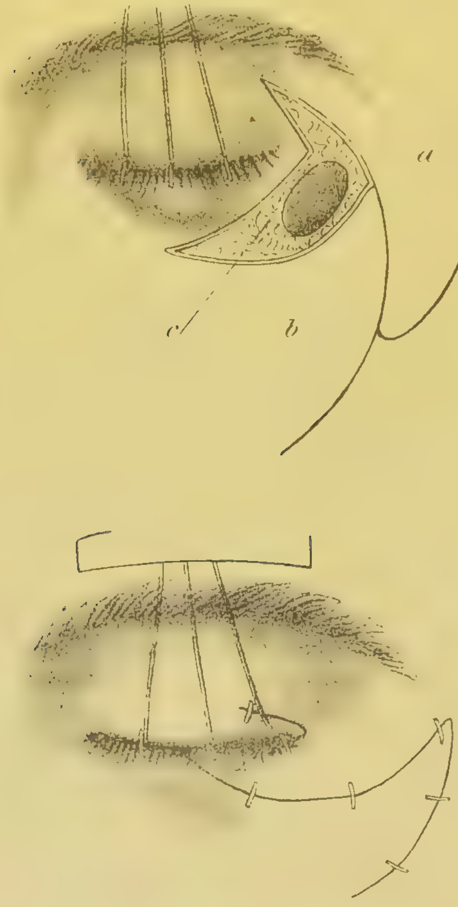

Fig. 180.—(After Masselon.)

taken. It should be transferred as soon as possible into the position it has to occupy. As union takes place principally by the opposed flat surfaces, a great number of stitches are not necessary. A few, however, should be introduced to keep the graft in position, and for this purpose three at least are required—one at each end, and one at the centre of its uppermost portion; in the lower lid, therefore, attaching it to the margin of the lid, in the upper lid to the skin. Before applying any dressing the skin graft should be raised, and the wound syringed or washed with boracic lotion, and all bleeding stopped. The graft should then be carefully applied to the raw surface below. The following dressing may be recommended:—Immediately over the graft is placed a piece of muslin smeared with iodoform ointment, over this a pad of cotton wool dipped in corrosive sublimate solution, and this again covered with a considerable quantity of dry cotton wool, the whole being kept down by means of a flannel or demet bandage. The dressing should not be changed for two or three days, and it is to avoid any difficulty in changing that it is advisable to have ointment next the graft, while the cotton wool and bandage secure the necessary immobility. The subsequent dressings do not require to be so carefully applied.

OPERATIONS FOR PTOSIS.

An operation, the object of which is to permit of the possibility of raising a drooping lid, must of course be performed in such a manner as to interfere as little as possible with the power of closing the lid. Removal of an elliptical fold of skin from the lid can consequently only be employed in cases where the ptosis is of moderate degree, and where there is a redundancy of skin, or at all events the amount of effect produced must be carefully regulated to admit of a sufficient degree of closing. In most cases one of the three operations about to be described will be found more suitable. The two first of these may be combined with the removal of skin to an extent suited to the requirements of any particular case.

Dransart's thread operation, the object of which is to bring the occipito-frontalis muscle to act on the lid through the medium of cicatricial bands, is performed as follows:—One end of a thread is passed below the skin from just above the upper border of the tarsus to a few millimetres above the eyebrow, the other end is carried in a similar manner parallel with the first. Similarly situated loops are introduced to either side of this one, these are then all knotted tightly over pieces of drainage tubing, the lid being thereby raised to an extent rather greater than it is desired to obtain permanently. The knots are tightened above from time to time, until the threads have ulcerated their way through the tracks along which they were passed. This operation is suitable for cases of incomplete ptosis, but is rather severe, as it is followed by a prolonged and painful swelling.

Eversbusch's Operation.—Where there is any independent power of the levator palpebræ, another operation, the principle of which is also to produce a shortening of its tendinous attachment to the tarsus, may be tried. Such a method of operating was recommended some years ago by Eversbusch, and it is by far the most satisfactory treatment of congenital ptosis, when not complete. I have performed this operation in more than a dozen cases, and have usually succeeded in effecting a considerable improvement. An incision occupying the whole breadth of the lid, as far as the clamp renders possible, is made in a horizontal direction, and equidistant between the lid margin and the eyebrow. The tissues are then dissected up or separated with the end of the handle of the bistoury to the extent of about a quarter of an inch, so as to expose the connective tissue, which springs forwards and is readily recognised. At the same time the lower border of the incision may be also slightly undermined. A strong piece of catgut with a curved needle at either end is then taken; the one needle is passed into this tendinous tissue as far up as possible, and brought out again a few millimetres from the point at which it was introduced. Both needles are then passed parallel to each other, and at two or three millimetres distance apart, or thereabout, below the skin and muscle of the lower portion of the lid, along the surface of the tarsus, and brought out at the free margin of the lid. Similar loops are next passed to the inner and outer sides of this central one. After this has been done the clamp is removed, the bleeding stopped to some extent, and the wound in the skin brought together with stitches. In the worst cases a piece of skin may be removed before doing this. Finally, the loops are knotted along the margin of the lid. Eversbusch recommends the use of glass beads slipped over the two ends of the thread before knotting, so as to prevent their cutting too deeply into the lid margin, but this is not necessary when catgut is used. After the completion of the operation both eyes should be bandaged, and kept so for several days, the bandage being changed each day. What actually takes place as a result of this proceeding is that the tendinous attachment of the levator is doubled down over the tarsus, where it forms fresh adhesions. Figs. 181 and 182, taken from Eversbusch's drawings, show one of the loops before and after it is knotted at the lid margin.

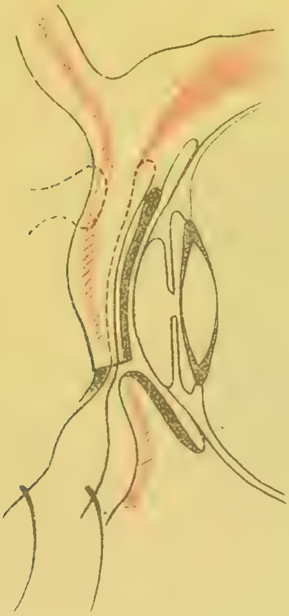

Fig. 181.

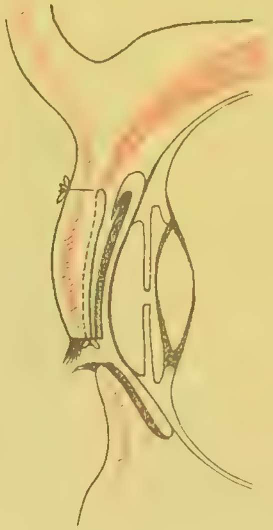

Fig. 182.

Snellen recommends a thread operation, by which the shortening of the tendon of the levator may be effected without cutting down upon it. Two or three parallel threads are carried by needles entered through the skin just above the tarsus, and passed below the mucous membrane as far up as possible, whence they are carried forward to underneath the skin near the eyebrow, and, finally, after being passed below the skin of the lid, are brought out at their points of entrance. The two ends of each loop are then knotted together, enclosing, amongst other tissues, the tendon which it is desired to shorten. I have performed this operation in six cases, and though it has led to some improvements, it has not in my hands proved as successful as the more radical operation by Eversbusch's method.

In some cases of congenital ptosis there appears to be no levator muscle at all, or it exists only in a very rudimentary condition. In such cases, and in sufficiently long-standing cases of complete paralytic ptosis, where an operation may be desirable, that recommended by Panas seems to offer the best chance of a successful result.

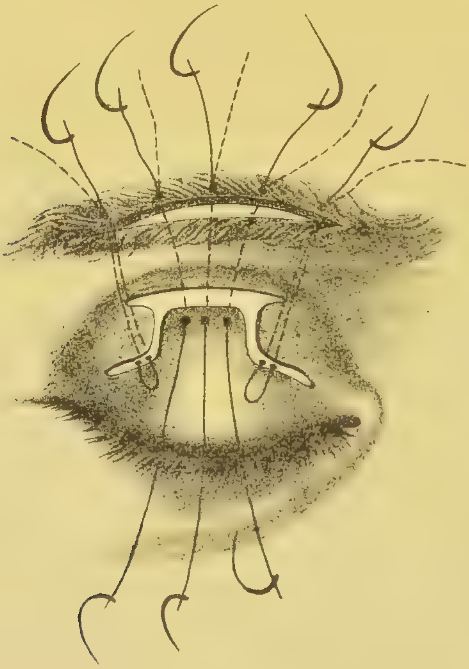

FIG. 183.—(After Panas.)

Panas' operation is performed in the following manner:—An assistant applies his hand to the patient's forehead to prevent the drawing down of the skin of the lid, by which the natural arrangement of the tissues would be interfered with, and the necessary incisions could not be made with sufficient precision. The operator then begins an incision along the line of the upper border of the tarsus. The incision is, however, not continued along the whole line, but, beginning immediately over one canthus, and leaving a central portion of a third of an inch uncut, ends at a similar point above the other canthus. A second horizontal incision, with a slight convexity upwards and not quite an inch in length, is made in the position of the fold separating the eyebrow and lid, and therefore just about over the orbital margin. This second incision must pass through all the tissues down to the periosteum. It is then joined by means of two short vertical

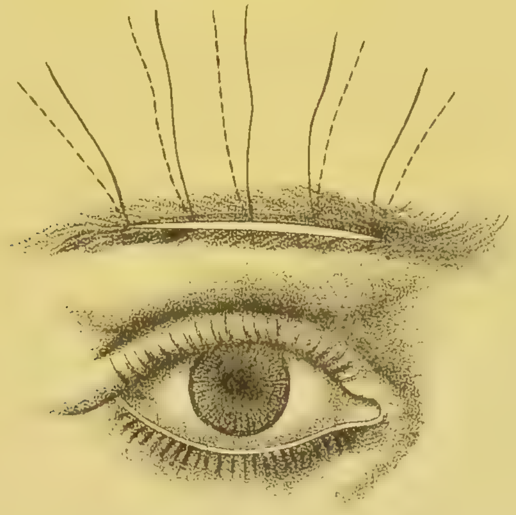

FIG. 184.—(After Panas.)

incisions with the inner extremity of the external portion and outer extremity of the internal portion of the lower incision. Yet another incision is made parallel to the second, and somewhat more than one inch in length, along the upper border of the eyebrow, and deep enough to extend to the periosteum. The little peninsula of skin and muscle marked out by the middle and lower horizontal and the two vertical incisions is next dissected free from the tarsus down to its ciliary border, care being taken not to interfere with the suspensory ligament of the lid. The bridge between the middle and upper incisions is then undermined, in doing which any wounding of the periosteum or suspensory ligament must be avoided. When this has been done, the dissected flap is pressed up underneath the undermined bridge, and attached by three sutures to the upper edge of the upper incision. Panas' operation results in a raising of the tarsal portion of the lid, and at the same time admits of the occipito-frontalis muscle to a great extent assuming the function of the absent or paralysed levator. The disfigurement caused by this operation is also extremely slight, while it in no way interferes with the closing of the lid. I have only had occasion to perform this operation twice, but am satisfied as to its efficiency.

OPERATIONS FOR SYMBLEPHARON.

The wound left after the detachment of the cicatricial tissue, by which the lid has been rendered adherent to the eye, may be covered in different ways. The method of operation to be selected in any particular case will depend on the extent of this denuded surface, as well as on the condition of the surrounding conjunctiva.

Where the cicatricial band is small and the rest of the conjunctiva healthy, the loss of substance may be made good by a transposition or glissement of the adjoining conjunctiva. The band of cicatricial tissue is grasped firmly with a pair of fixation forceps, and put on the stretch. Its attachment to the cornea is then carefully dissected off with a small bistoury, and the freeing of the lid completed by snipping the other attachments with scissors. The conjunctiva to either side is next undermined to an extent which permits of the edges being readily brought together by sutures. The stitching should be carefully performed, and as many stitches as possible used, particular attention being paid to the covering of the lowest part. Where the cicatricial band is of some size, it may be turned back and made use of as a partial covering for the raw surface of the inner side of the lid. This may be done by a loop-stitch passed through its apex, then through the thickness of the lid, on the skin surface of which it is tied over a piece of drainage tubing. This latter proceeding is seldom of much use; it is generally better after cutting away the redundant cicatricial tissue to rely on the frequent use of oil during the cicatrisation of the lid.

Another operation which is suited to the same class of cases, and by which perhaps a rather large defect can be covered, is *Teale's transplantation operation.* By this operation two flaps of conjunctiva are taken from either side, one of which is used for covering the inner surface of the lid, while the other serves as a covering for the eye. The flaps are cut as represented in Fig. 185, which is copied from a drawing in Mr. Swanzy's *Handbook of Eye Diseases,* altered from his original one by Mr. Teale himself.

The complete transplantation, *or grafting of mucous membrane,* has lately taken the place of other operations for symblepharon. Such a method of operating is certainly applicable to cases which would otherwise not admit of interference. The mucous membrane may be conveniently taken from the mouth, and should be half as large again as the defect which it is to cover, and devoid of any submucous tissue. Owing to the tendency which it has to curl up, it is impossible to get it into good position without the use of a number of stitches. In order, too, to ensure its applying itself to the wound throughout, it is well to put in one or two loop-stitches as well at the fornix, which are brought through the substance of the lid and tied at the outside. The graft should not be definitely fixed until all bleeding has stopped; it is therefore best to begin with the stitches that attach it to the lid.

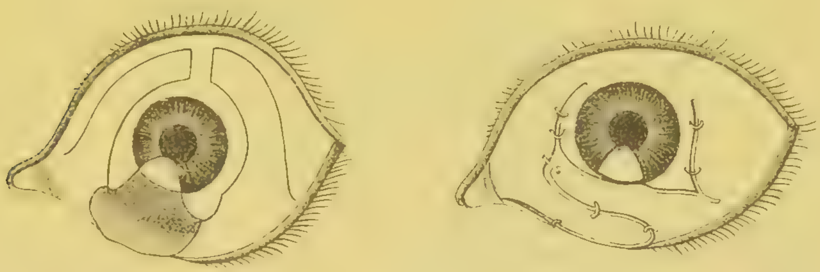

FIG. 185.

When there is a redundancy of skin in the lower lid, a piece of skin from this situation may be conveniently used instead of mucous membrane, the character of which it to a great extent slowly acquires. It is much more easily fixed in position, and does not require so many stitches. Snellen has recently recommended an operation for bad cases of symblepharon, which consists in clothing the inner raw surface of the lid, after it has been detached from the eye, with a flap of skin dissected from the temple in the immediate neighbourhood of the outer angle of the lids. After cutting the elongated flap, an opening is made below its base into the conjunctival sac. The skin is then pushed through this opening, so that its raw surface comes to lie against that of the detached lid, and stitches are introduced to maintain it in this position. I have performed this operation several times, and obtained very satisfactory results.

OPERATIONS ON THE PARTS CONNECTED WITH THE SECRETION AND EXCRETION OF THE TEARS.

Excision of the Lachrymal Gland.—An incision is made in the skin, over and in line with the upper and outer margin of the orbit, immediately below the eyebrow, and extended in depth until the hypertrophied gland or tumour comes into view. The edges of the wounds thus made are held apart with hooks by an assistant, and the gland drawn forward by means of another sharp hook and carefully dissected out as far as possible. When this incision does not afford sufficient room for the removal of the gland, a larger opening may be got by dividing the outer canthus by means of a horizontal incision, carried far enough to meet the other at a point. This, which was first recommended by Lawrence, is preferable to extending the incision inwards, as that would endanger the force of opening of the lid by interfering too much with its suspensory ligament.

Bowman's Operation for Fistula of the Lachrymal Gland.—The object of this operation is to establish a fistula on the conjunctival surface, and thus permit of the opening in the skin being permanently closed. A silk thread armed with a needle at either end is used. The first needle is passed into the fistulous opening, carried up it for a short distance, and then made to pierce the tarsus and conjunctiva. The thread is then drawn out at the inner surface of the lid. The other needle is introduced in the same way, but brought out about quarter of an inch from the first and a little nearer the lid margin. The two ends are then secured by being plastered to the skin to the outer side of the palpebral aperture. They are left in this position for ten days or a fortnight, after which the external opening is closed by paring its edges and stitching.

Slitting the Canaliculi.—This little operation was first practised by Bowman, and is now always performed with the narrow probe-pointed knife introduced by Weber. The probe-pointed end of the knife is inserted into the punctum lachrymale (see Fig. 186). The skin, and through it the inner portion of the lid margin, is then put on the stretch with the thumb of the hand, care being taken to neither invert or evert the lid to any extent. After this has been done, the knife is brought into such a position as to be in a line with the canaliculus as far as possible, whilst its cutting edge is directed upwards and slightly inwards. It is then passed along the canaliculus till its point touches the inner wall of the sac and presses it against the nose. A quick movement round this point as centre is then given to it, upwards if the lower canaliculus is being slit, and downwards in the case of the upper canaliculus. In this way the narrow bridge forming the roof or the floor of the canaliculus is severed up to its entrance into the sac. If the slitting is performed for dacryocystitis, the knife should be passed on into the sac, and, with the cutting edge directed forwards, a slight sawing movement made. The slit canaliculus shows very little tendency to heal up, as union takes place between the skin and mucous membrane to either side of the cut. It is advisable, however, to pass a probe along it after twenty-four hours, so as to ensure its remaining patent. When

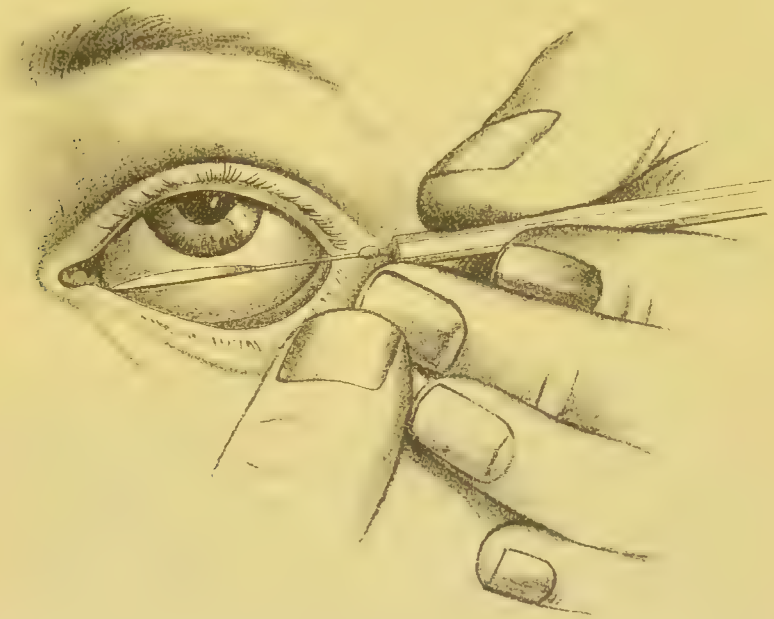

FIG. 186.

probing of the duct is necessary, its direction, as explained in Fig. 19, must be borne in mind.

Extirpation of the Lachrymal Sac is by no means an easy operation, owing to the deep position occupied by the mucous membrane which has to be removed, and the great vascularity of the parts. The incision made through the skin in the line of the sac should be sufficiently long to enable one to get well at the sac; it may therefore be carried down as far as required for the purpose. When the mucous membrane is exposed, the lips of the external wound should be forcibly held aside with sharp hooks. The mucous membrane is then seized with a pair of toothed forceps, and its removal begun by dissecting it away from the overlying tissues. Often it is so soft that it can only be got away piece-meal,, and then gives more difficulty owing to the bleeding. A catgut drain and a good firm pad and bandage are afterwards applied.

Destruction of the lachrymal sac with the thermocautery may be performed in the same way. It is best, when possible, to pass a probe into the sac from above before making the incision, which need not be quite so long as for extirpation. The probe also serves as a guide to the extent of the cauterising required. I have found a fine curved platinum point, such as that used for septic ulcers of the cornea, the most suitable.

OPERATIONS ON THE CONJUNCTIVA.

Operation for Pterygium.—The pterygium is seized with a pair of forceps held in the one hand, and the portion adherent to the cornea carefully dissected off with a small bistoury or Sichel's knife. When this is done an incision is made from the corneo-scleral margin at the upper edge of the pterygium with a pair of straight scissors, in the direction of and extending to the centre of its base. A similar snip is made from the lower margin of the pterygium at the border of the cornea, meeting the first at a point. In this manner a lozenge-shaped piece is removed. Then there remains a raw surface in the conjunctiva, which is to be covered by undermining the conjunctiva above and below, and drawing it together with stitches.

The operation of Peritomy or Syndectomy is performed with fixation forceps and a pair of scissors, a portion of the conjunctiva immediately surrounding the cornea, a quarter of an inch or so in breadth, being seized with the forceps, and snipped off with the scissors as close as possible to the cornea. The band-shaped portion thus removed may extend all round the cornea, or only in part, the latter being in most cases sufficient.

OPERATIONS ON THE CORNEA.

Operations for Corneal Staphyloma.—When a partial staphyloma of the cornea becomes unsightly, it may be reduced in size by the following simple operation. A cataract needle is introduced through its base, and held in one hand. An elliptical piece of the cicatricial tissue of which the staphyloma is composed is then cut out by making one incision at the one side of the needle with a narrow cataract knife, and another from the other side converging towards the first, and in such a manner that the portion held by the needle, and consequently the needle itself, is cut out. This can be done very quickly. Antiseptic precautions should be taken and a firm bandage applied.

In cases of more complete staphyloma, attempts have been made to retain a better stump by covering up the wound resulting from the removal of the protruding cicatricial tissue which takes the place of the cornea. Inasmuch, however, as if inflammation be set up there is considerable risk of sympathetic ophthalmitis, it is rarely advisable to attempt an operation of this kind. Critchett was the first to recognise the importance of closing up the wound, and this he did by inserting a number of deep stitches through the sclera before removing the staphyloma. The needles were entered through the sclera above the protrusion, carried underneath it, and brought out again through the sclera below it. The sutures could then be drawn through and tied as soon as the corneal cicatricial tissue was removed. This method of operating was afterwards modified by Knapp, who used conjunctival sutures instead of scleral ones. More recently the following operation has been recom-

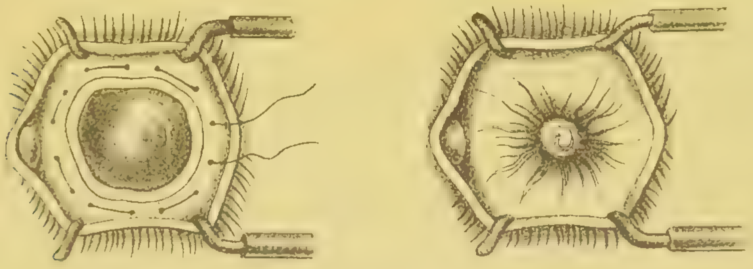

FIG. 187.—(After Wecker.)

mended by Wecker, and is probably the best of its kind. Before removing the staphylomatous protrusion, the conjunctiva and subconjunctival tissue surrounding the cornea is freely undermined for some distance. A strong suture is then run through this loosened tissue at a distance of a quarter of an inch or so from the corneal margin, and with not more than a quarter of an inch intervening between each puncture made by the needle until the other end of the thread is brought out near to the first puncture. The protrusion is then cut off by transfixing it through the middle, and cutting outwards, then seizing the end of the flap thus formed and removing the rest with scissors. As soon as this has been done the two ends of the continuous suture are drawn together and firmly knotted (see Fig. 187). The effect of this is to draw the conjunctiva over the wound, and thus prevent the escape of the vitreous; the lens generally escapes with the removal of the protrusion; if it does not, the capsule may be scratched so as to permit of its escape. It is of the utmost importance that antiseptic precautions should be taken in this operation. The thread may be allowed to remain for a week at least. If it cuts through at any part, a fresh stitch may be used for bringing the conjunctiva together where this takes place.

The most suitable operation for cases of complete staphyloma is evisceration of the globe (see page 703).

Tattooing of the Cornea.—In cases of dense leucoma without staphyloma the appearance of the cornea may be improved by tattooing. The same proceeding may also be employed for the improvement of vision which is often greatly interfered with by a smaller nebula covering the pupil more or less completely. If the pupil be completely covered, this may be combined with the formation of an artificial pupil where there is enough clear cornea surrounding the nebula. If on the other hand a portion, even a small portion, of the pupil remains uncovered, tattooing alone is likely to be sufficient. Tattooing is best performed with a number of sewing needles held together, either in a special holder or by being embedded in sealing-wax. The points of the needles should be all on the same level. After the cornea has been rendered insensitive by means of cocaine, some Chinese ink rubbed down in water into a thin paste is applied with a camel's-hair brush in as thick a layer as possible to the surface of the leucoma, and dabbed into it by repeated stabs with the bundle of needles. Some operators use a single grooved needle which they insert very obliquely into the cicatrised cornea. In any case the pricks should be made obliquely and not perpendicular to the surface. When the tattooing is performed for a small central nebula, it is better to use the needles first, and afterwards rub in the ink. In this way it is possible to make the edges of the staining coincide with those of the nebula. The eye must be steadied all the while as far as possible with the finger, not fixed with forceps, as the ink, getting into the wound made by the teeth, would leave a stain. After the stabbing process has been repeated a good many times, with the addition when necessary of fresh ink, the excess is washed away with some corrosive sublimate lotion. The necessary degree of blackness may be obtained at one sitting; but it is better to repeat the process once or twice.

Operation of transplanting Clear Cornea.—The numerous attempts which have been made to graft portions of clear cornea so as to take the position of the intransparent cicatricial tissue constituting the leucoma have been all more or less unsuccessful, owing to the loss of transparency, which sooner or later befalls the graft even where it has retained its vitality. Von Hippel has recommended an operation which, in his hands, appears to have been attended by a very encouraging degree of success. The principle of this operation is to remove by means of a trephine a circular portion of the leucoma, leaving, however, the posterior elastic lamina of the cornea intact. This appears to be an essential element in the process of the operation, and von Hippel was led to adopt it in consequence of the result of Leber's experiments on the secretion and excretion of the intra-ocular fluids, by which it was demonstrated that the transparency of the cornea depended on the integrity of the membrane of Descemet. The trephine used for the purpose has a diameter of 4 millimetres, and is made to execute a number of revolutions round its axis by means of clock-work, which can be set in motion by a slight touch with the fingers on a button. This circular movement is arrested, too, as soon as the pressure on the button is stopped. Immediately above the cutting edge is a small projecting ledge which can be screwed into any position required, and which prevents the trephine passing too deeply into the cornea. The portion marked out by the circular cut is carefully removed with knife and forceps. A piece of the whole thickness of a rabbit's cornea is then excised with the same trephine and immediately transplanted. Care must be taken that the trephined portion has in each case perpendicular sides, and is not funnel-shaped, as this would interfere with the proper adjustment of the graft. The cornea is afterwards covered with iodoform and a bandage applied. This operation is only suitable for cases of complete leucoma, where there is no adhesion of the iris to the cicatricial tissue. It is of no use in cases of leucoma adherens.

OPERATIONS FOR CONICAL CORNEA.

All the operations for conical cornea are unsatisfactory, in so far as it seems almost impossible to predict what will be their effect. Von Graefe attempted to induce a cicatricial contraction in the neighbourhood of the apex of the cone, with the object of bringing about a more normal curvature of the cornea. *Von Graefe's operation* consists in removing a small portion of the cornea with a cataract knife in the immediate vicinity of the apex of the cone, taking care not to perforate the cornea. The wound thus formed is converted into an ulcer by being touched four or five times at intervals of three days with a solid piece of mitigated caustic. Afterwards the anterior chamber is punctured through the base of the ulcer, and this repeated three or four times every other day, after which the ulcer is allowed to heal. A small iridectomy is then made opposite the clearest part of the cornea. Cauterisations with the thermo- or electro-cautery have now to a great extent taken the place of von Graefe's method of producing a corneal ulcer. They are certainly safer and more easily regulated in their effect. Probably in most cases it is more the exclusion of the rays, which otherwise pass through the central portion of the cornea by the establishment of a dense leucoma in this situation, which gives rise to any improvement of vision, than any alteration of curvature which may be effected at the same time.

Bowman's Trephine Operation.—With a small trephine a circular portion, not greater, as a rule, than 3 millimetres in diameter, is removed from the apex of the cone. The trephine is not allowed to penetrate the whole thickness of the cornea, as, if a complete disc be removed, the operation is almost invariably followed by an anterior synechia. After, therefore, circumscribing the portion to be cut out to a depth which is considered safe in any case, generally about $\frac{3}{4}$ of a millimetre, it is removed with knife and forceps, and the base punctured to admit of the escape of the aqueous.

Bader's operation consists in removing an elliptical piece of the cornea $1\frac{1}{2}$ to 2 millimetres in breadth at the broadest part from the apex of the cone, and then applying a tight bandage. This operation is also liable to be followed by anterior synechia, but may give very good results.

Saemisch's section of the Cornea for Hypopyon Ulcer.—This is performed in the following way:—A narrow Graefe's knife is passed at the one side of the corneal ulcer into the anterior chamber, the cutting edge of the knife being directed forwards. A counter puncture is made in the sound corneal tissue immediately to the opposite side of the ulcer and the intervening tissue, that is, the base of the ulcer, divided by cutting outwards. In the case of large ulcers the wound then formed is pretty extensive, and is apt to be followed by disagreeable results—prolapse of iris, synechia, staphyloma, &c. I have always found that the effect of the operation is obtained with equal certainty by merely dividing in the same manner that portion of the base which is infiltrated. Often the wound has to be reopened several times before healing begins to take place.

The operation is now very seldom required if the thermo-cautery be properly used. (See p. 131.)

The removal of foreign bodies from the cornea presents greater or less difficulties, according to the depths at which they lie. In the great majority of cases they are quite superficial, and are then easily removed with the corneal spud. The surgeon should stand behind the patient, whose head is allowed to rest against the operator's chest. The lids are held apart with the fore and middle fingers of the left hand, and by gentle pressure against the margins, so as to avoid everting them. With the fingers in this position a certain amount of steadying of the eye is rendered possible at the same time. The spud should be inserted immediately under the foreign body, great care being taken not to injure any other part of the cornea by scraping the epithelium unnecessarily. Any digging that is required must be confined to the part at which the foreign body is lodged. When it is very deeply embedded in the cornea, it may be necessary to get at it by making use of a sharp needle. Care must be taken not to push the foreign body deeper; when, therefore, it has been well freed, the spud must be used and pushed well under it.

Nuël's operation for ruptures at the Corneo-scleral Juncture.—The cases for which this operation is applicable are referred to at page 150. It consists in making a section with a narrow knife, which reopens the old wound in the sclera, and then drawing the surrounding conjunctiva over this opening by means of a special suture. The object of the operation is to cover the wound with as thick a layer of superficial tissue as possible, and thus permit of the proper re-establishment of the anterior chamber. To effect this the knife, after cutting through the deep cicatricial tissue filling up the space between the lips of the wound in the sclera, is directed backwards, so as to cut out a deep flap of conjunctiva. A conjunctival suture is then introduced in the following manner:—It is entered at the equator of the eye, as far back as possible, and passed out and in, or run through the conjunctiva for a considerable distance, parallel with the corneo-scleral margin. The needle is then carried diagonally over to the conjunctiva immediately surrounding the cornea at the opposite end of the wound, and the thread run in a similar manner close to the cornea, and finally brought out beyond the wound at the other side. The two ends of the thread are then tied tightly together. In this way a large mass of conjunctiva is puckered up over the wound, in a much more efficient manner than could be done by the introduction of a number of sutures in the ordinary way.

IRIDECTOMY.

The various conditions for which the performance of an iridectomy, *i.e.*, the excision of a portion of the iris, may be necessary, have been referred to in the preceding chapters. Some differences in the method of performing this operation require attention, according to the object for which it is employed.

Iridectomy performed for merely optical reasons should, as a general rule, be small, and it is not necessary to remove the portion of iris up to its peripheral attachment. If the case be one of dense nebula of the cornea, the portion of iris removed, or the "artificial pupil," should be opposite the clearest and most normally curved portion of the cornea. This portion must, besides, be not too peripheral; that is, besides transparency and curvature, the radial extent of the transparent portion has to be taken into consideration. Other conditions being equal, the best positions for an artificial pupil for optical purposes are inwards, or inwards and downwards. Where, on the other hand, the case is one of partial cataract, and the object of the operation is to admit the rays of light through the clear peripheral portions of the lens, it is advisable always to perform the iridectomy upwards. The reason of this is that there is always a possibility of the cataract becoming more complete at some future date, and necessitating extraction. When this does happen, the artificial coloboma upwards does not interfere in any way with the operation.

Another class of cases where an iridectomy is required for optical purposes is where the pupil has been closed and bound down by synechiæ, as the result of iritis. In such cases the choice for the position of the iridectomy should be regulated by the condition of the pupil, as far as that can be made out by careful inspection, if need be, after the use of a mydriatic. The excision of the iris should be made where the pupil is least bound down. Other things being equal, it is best to make it upwards. The reason of this is that the operation in such cases, though mainly performed, it may be, for optical purposes, should be undertaken with the possibility kept in view of some future recurrence of the inflammation taking place, so that the coloboma should be made of a good size. The removal of a portion of iris, again, which is dragged on owing to the existence of an anterior synechia must be sufficiently large to entirely free the unattached portion. In many cases of this kind there is no necessity for any operation at all; but where there is distinct irritation produced, it should not be delayed. Often, indeed, it is advisable to free the iris on both sides of the adhesion. This may be done by a double iridectomy, performed either at the same time, or, more easily, at an interval of a few days.

The rules for the performance of iridectomy for glaucoma are specially referred to further on.

The instruments required for an optical iridectomy are—a spring speculum, a pair of fixation forceps, a bent triangular lance-shaped knife (often called a keratome), a pair of iris forceps, or iris hook (Tyrrell's hook), a pair of iris scissors, and a small flexible caoutchouc or tortoise-shell spud (see Fig. 188.)

In making the necessary section with the keratome, the operator may either push the point away from him or towards him; in performing an iridectomy upwards, therefore, he will in the first case stand behind the patient's head, and in the second at the side of the patient, and at the same time as much in front of him as possible. When there is plenty of room, that is to say, where there is a good anterior chamber, either manner of introducing the knife is equally easy. In introducing the knife, however, into a narrow chamber, the second is decidedly the better, as it enables the operator to watch the point much more closely. After the eye has been

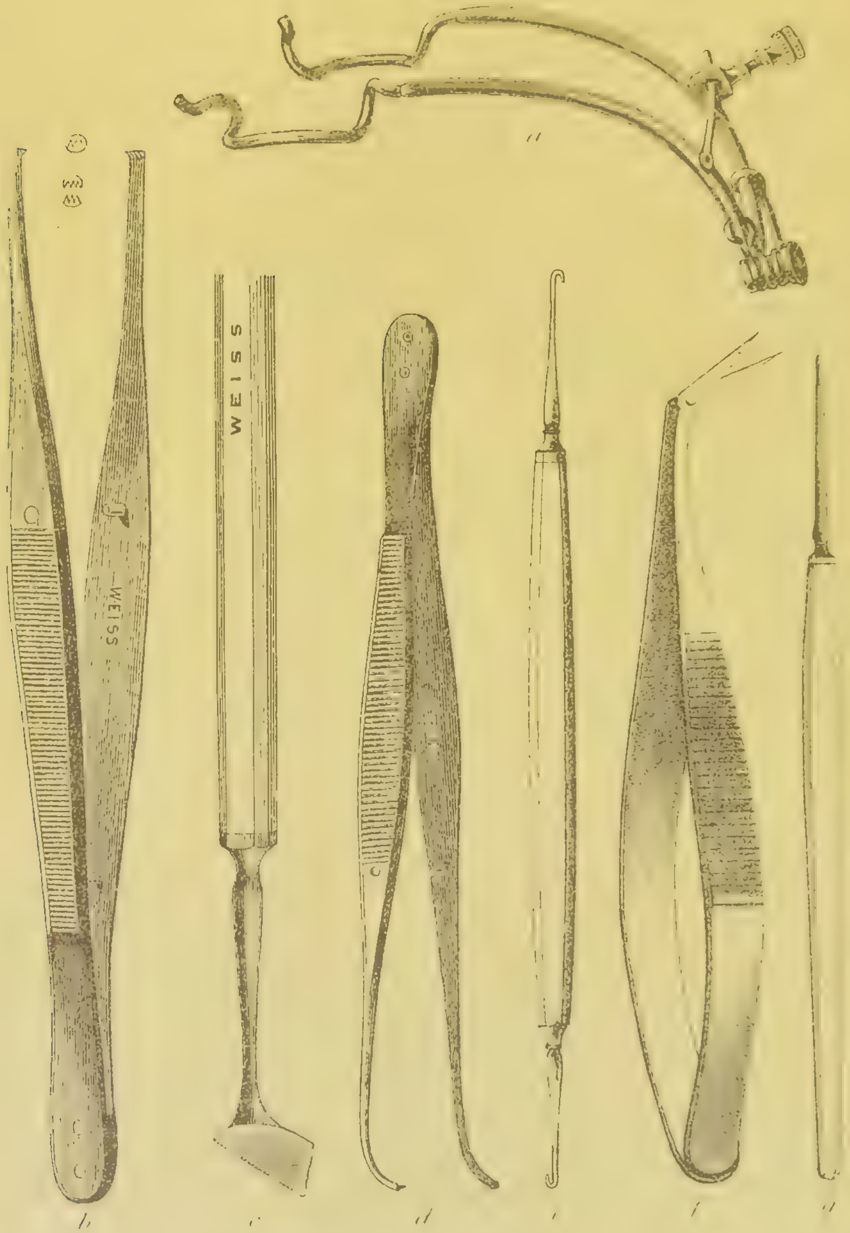

Fig. 188.—*a*. Speculum ; *b*. Fixation forceps ; *c*. Keratome ; *d*. Iris forceps ; *e*. Iris hook ; *f*. Iris scissors (M'Clure) ; *g*. Caoutchouc spud.

cocainised, and the conjunctival sac well washed out with the corrosive sublimate solution, the speculum is introduced, and the surgeon, taking the fixation forceps in the left hand, takes a firm hold of the conjunctiva and subconjunctival tissue at the opposite end of the diameter in which he intends to make the iridectomy. The knife should then be introduced at the apparent corneo-scleral margin, the point being directed at right angles to the surface of the cornea in that situation, and pushed forwards until it has just pierced the thickness of the cornea. The knife is then directed more forwards by depressing the handle, and, with the plane of the blade parallel with the iris, pushed rapidly *and without any hesitation* on into the anterior chamber, until the external wound has attained a sufficient size, say 4 to 5 millimetres for most optical purposes. The blade is then *slowly* withdrawn with the point tilted slightly more forwards. Tyrrell's hook, or the iris forceps, is then introduced into the anterior chamber. Where the operation is done for the displacement of the pupil opposite a clearer portion of cornea the hook is generally the best. It should be of platinum, so that it can readly be bent into any shape required. The point should be blunt and perfectly smooth. It is introduced with the hooked end on the flat, passed inwards parallel with the iris, but without touching it, until its end has got beyond the edge of the pupil; a slight rotation is then given to the instrument, so as to direct its point towards the lens, and it is slowly withdrawn until it catches well on to the pupillary margin. The iris can then be dragged out of the wound with the hook kept as flat as is consistent with its retaining a good hold of it. It should be slowly dragged out, and without any great traction, and then snipped off with the scissors. With a good assistant it is better for the operator to entrust the scissors to him and maintain fixation himself, with the forceps held in the other hand. Various forms of iris scissors are in use, but that figured will be found as good as any, and less likely to get out of order. When the iris forceps is used it should be introduced closed, until the points lie just over the margin of the pupil, when they are allowed to open, and are at the same time pressed gently down upon the iris. In this way a portion of the iris, including a portion of the pupillary margin, rises in between the two limbs of the forceps, which are then firmly closed and slowly withdrawn, until a good snip can be got with the scissors outside the wound, which leaves a piece of detached iris in the forceps. A stream of sublimate lotion may now be poured upon the wound. The eye is then closed and gentle friction made over the lid; in this way any part of the iris caught in the wound is as a rule liberated, but if the iris should not have returned to its place it may be gently dragged away, by passing the caoutchouc spud into the angles of the wound and on to the surface of the iris. This must only be done, and with great care, when the first proceeding fails. If there is any bleeding, the upper lip of the wound should be slightly depressed, so as to permit of its escape, but attempts to remove blood should not be persisted in for any length of time. The bleeding usually comes from the conjunctiva, a portion of which may have been snipped off along with the iris. Care should therefore be taken in using the scissors to see that only iris is included between its blades. A dressing is applied in the way described at page 689, and kept on for twenty-four hours, when it is reapplied as before. After forty-eight hours it may be removed altogether, and a shade worn over both eyes.

Iridectomy for Glaucoma.—It is impossible in the way already described to remove a portion of the iris which extends quite to its peripheral attachment. Unless it be actually torn away, a peripheral piece remains, corresponding in depth to the distance separating the iris periphery from the inner incision, plus the thickness of the cornea. In operating for glaucoma, the iridectomy should be large and also as peripheral as possible. Whether this be always, or even ever, absolutely necessary, may be open to question, but at all events such are the traditional requirements for the operation in that disease. The instruments required are the same as for the performance of an optical iridectomy, only the keratome should be larger, in fact as large as possible, and the iris forceps, and not the hook, used for seizing hold of the iris.

Some operators make the incision in cases of glaucoma with a narrow cataract knife instead of with a keratome. With such an instrument, with which the section is made from within and not from without, it is impossible to make as clean a section as with a keratome, if an attempt be made to make it very peripheral. The difficulty is increased, too, if the anterior chamber be very narrow, as is frequently the case; indeed it is only with a keratome that it is possible to make a clean peripheral incision into a narrow chamber. The cataract knife, when used, presses the iris in front of it, bruising it, or even detaching it in part from its peripheral attachment.

Glaucomatous eyes are often small and deeply placed in the orbit. Owing to this, and to the greater size of the incision required in cases of glaucoma, and the consequent difficult and often painful nature of the operation, it is often advisable to anæsthetise the patient. If an anæsthetic be not used, great care must be taken to keep the patient's head perfectly quiet. Cocaine very often produces comparatively little local anæsthesia, though by its use the incision may generally be made without causing any great pain. Much depends of course on the state of the eye at the time of operation; in very acute cases an anæsthetic is almost absolutely necessary to avoid running any risks.

The keratome is introduced in the same manner as has just been described, but rather further back, about one millimetre behind the apparent sclero-corneal margin. Here, too, it is still more essential that the wound through the cornea should be direct and not sloping. After the point has penetrated, and the blade then turned somewhat forwards, the more rapidly it can be introduced into the anterior chamber the easier is it to get a sufficiently large incision. In withdrawing it, the one edge should be tilted very slightly sideways, and made to enlarge the opening to that side. This should be done leisurely, without any great haste; nothing looks uglier than to see a keratome rapidly jerked out of the eye after the incision has been made. The iris is then grasped with the forceps, which should be opened widely, so as to get hold of a good large piece. This is drawn out of the wound and cut as close as possible to it, with the object of cutting across a very peripheral part. Great care must afterwards be taken to prevent any encleisis, as it is of importance to obtain healing without any irritation, and eventually a smooth even cicatrix. If the anterior chamber does not re-form within forty-eight hours, it is best to leave the bandage off altogether, or at all events only apply it very lightly. In many cases it is useful to continue using eserine or pilocarpine drops for a week or a fortnight after the operation.

SCLEROTOMY.

An opening made into the anterior chamber as far back, or as nearly coinciding with its angle as possible, has received the name of sclerotomy, as the incision lies mainly in the sclera. An incision of this kind may be made in several ways. The plan which seems to be most commonly adopted is the following:—After having obtained a maximal degree of myosis with the use of eserine or pilocarpine drops, a narrow cataract knife is introduced at a point in or slightly above the horizontal diameter of the cornea, and quite one millimetre from its margin. It is carried across the anterior chamber to a point exactly on the same level on the other side, where a counter puncture is made. Then, with a slight sawing movement, an incision is begun, which, if finished, would separate the whole of the cornea above the points of entrance and exit of the blade from its connection with the sclera. Only two-thirds, however, of this section is to be completed, one-third of which is cut with the portion of the blade next the point, and the other third with that next the handle. A bridge of tissue equal to either incision is left. The knife is then carefully withdrawn, and any prolapse of iris replaced with the caoutchouc spud. The use of eserine must be continued during the healing process.

Another method consists in making a smaller incision with the same knife, and while finishing the section throughout as far as the sclera is concerned, leaving as much of the conjunctiva uncut as possible.

Yet another method, and in my experience the best, is to make the incision with a large keratome, in every way as for a glaucoma iridectomy. It is generally possible, in cases which are suitable for sclerotomy, to prevent prolapse after this incision by the previous use of eserine, and, if necessary, the use at the time of the caoutchouc spud. If, however, efforts in this direction fail, the operation can be converted into a good iridectomy by the excision of a portion of iris. By the other methods any prolapse of iris cannot be so satisfactorily treated. Such operations are, besides, if the incision be made really peripheral, subject to the same practical objections as have been urged against the use of a cataract knife in the performance of iridectomy.

OPERATION FOR PROLAPSED IRIS.

If the patient be seen shortly after the occurrence of the perforation which has led to the prolapse, the iris may be either replaced or seized hold of with the iris forceps and snipped off. The treatment in any particular case is mentioned at p. 149. When some time has elapsed since the prolapse took place, an operation may be required for removing it, so as to obtain a smooth cicatrix. This is best performed as follows:—The centre of the cicatricial tissue, including the prolapsed iris, is transfixed with a narrow cataract knife, which is then made to cut outwards, so as to separate the base of the prolapse from the underlying cornea. The detached portion is next seized with a pair of iris forceps, and snipped off along with the remaining prominence. If an attempt be made to cut it away without first using the knife, the scissors will be found not to get a good hold, and glide over it, so that in that way it is generally impossible to remove it sufficiently thoroughly.

CATARACT OPERATIONS.

Discission or *needling* for cataract is practised in the case of lenticular opacities in young individuals. The object of the operation is to allow the aqueous humour to come in contact with the lens substance, which is thus softened and slowly absorbed. To effect this, it is necessary to make an opening in the lens capsule, and at the same time break down, to some extent, the substance of the lens as well. The instrument used for this purpose is a sharp needle, the body of which is made almost imperceptibly tapering, and at the same time accurately rounded, so as to prevent any escape of aqueous whilst it is in use. Before operating, the pupil should be well dilated with atropine. Having taken a good hold of the conjunctiva with the fixation forceps held in the left hand, the needle, which has been rendered thoroughly aseptic, is pushed through a peripheral portion of the cornea until it reaches the capsule of the lens. The handle is then slightly depressed, and at the same time the needle pushed a little further through the cornea, and then, by a movement round the portion of it which is grasped by the cornea as axis, it is made to make a linear cut through the capsule in a vertical or nearly vertical direction. It is then slightly withdrawn, and a horizontal incision made through the capsule in the same way. The capsule is thus opened by a crucial incision, and after this has been done, more or less of the lens may be stirred up with the end of the needle, care being taken that the whole thickness is not pierced at any place. The needle is then quickly withdrawn, the eye washed with a stream of corrosive sublimate solution, and a bandage applied. For some time afterwards the pupil must be kept well dilated with atropine, so as to prevent any synechiæ forming.

The main point in the operation consists in making a satisfactory opening in the capsule. If the needle be merely stuck into the lens, and the point then moved about so as to stir it up, a proper absorption does not follow, and there is a risk besides of rapid swelling taking place within the capsule, which may give rise to considerable irritation.

In children no further operation is required as a rule. Every case should, however, be carefully watched for some days afterwards, as it occasionally happens that when a considerable quantity of softened and swollen opaque lens matter falls all at once into the anterior chamber, it interferes with the normal excretion of the fluids of the eye, and may thus give rise to glaucomatous symptoms, which may endanger the eye. When this occurs, it is often at the same time accompanied by pain and vomiting. It is the more likely to occur the older the individual on whom the operation is performed, and the more freely the capsule and lens have been needled. Under these circumstances it is necessary to extract as much of the lens matter as can be readily got away from the anterior chamber. There are different ways of doing this, but the best is by making a small *linear extraction.* This is done with a keratome. The keratome is entered through the cornea, about 2 millimetres from its margin, taking care that the wound is quite perpendicular to the surface. The incision thus made should be 4 or 5 millimetres in length. After withdrawing the keratome, the upper lip of the wound is depressed with a small scoop, along which the aqueous and lens matter are allowed to escape. If the whole of the contents of the anterior chamber do not come at once in this way, the eye may be closed for a minute or two, and the manœuvre repeated. It is best, if possible, to avoid introducing the scoop into the chamber, and in any case it is not necessary, although of course it hastens matters, to extract all the lens substance.

Some surgeons make a practice of always extracting a few days after needling, whether that be followed by any irritation or not. The time required for the cure of the cataract is thus shortened, and, if a linear extraction performed in the way described be practised, with proper antiseptic precautions, it is a very safe proceeding. There is necessarily, however, a slight increase in the risk as compared with needling alone and leaving the absorption to take place in the course of nature.

The broken-up lens matter may also be removed by the method of *suction*. Having made a linear incision in the cornea in the manner already described, the nozzle of a suction syringe is introduced well into the anterior chamber, and the softened lens matter slowly sucked up into it. Two forms of syringe are used, Bowman's and Teale's. In the one the suction is got by means of raising a piston with the thumb, while the tube of the syringe is firmly held by the first and second finger inserted into two rings fixed to the tube. The other suction curette consists of a silver nozzle attached to a glass tube four or five inches long, ending in a flexible indiarubber tube, with a glass mouthpiece at the other end. The suction is made with the mouth. Those who attempt removing lens matter by suction should pay attention—first, to securing the absolute asepticity of the syringe; and, secondly, to the manner in which they hold it in the chamber. An instrument of this kind, it must be remembered, is more difficult to render aseptic than most others. The nozzle has its opening on its anterior surface, consequently it is necessary, in order to avoid having to exert too powerful a suction, to get it well behind the matter which it is desired to suck up. Care must also be taken that it does not get behind the iris, or be allowed to enter so deeply that the posterior lens capsule is ruptured. Very pretty results may be obtained by this method; but it is very questionable whether on the whole it is better than the simple linear extraction, and it is certainly not so safe.

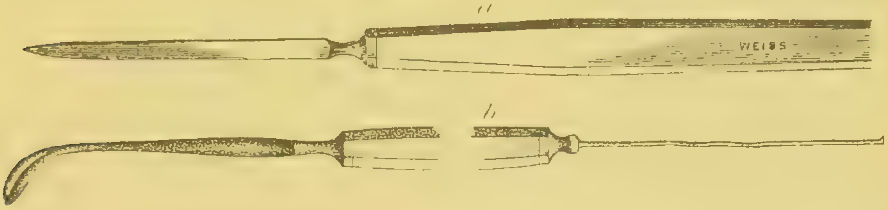

FIG. 189.—*a*. Graefe's knife; *b*. Cystotome and tortoise-shell scoop.

Cataract Extraction.—The method of extracting senile cataract now almost universally followed is one which differs but to a slight extent from that introduced by von Graefe as his modified linear operation. Slight differences made by different operators in the position of the incision have led them to describe modifications, which they have been pleased to dignify by the name of new methods, but these have in almost all cases been so trivial that they do not require any serious consideration.

The instruments required for extraction with iridectomy are the following (see Figs. 188 and 189):—A spring speculum, a fixation forceps, a narrow cataract knife (Graefe's knife), a pair of iris forceps, iris scissors, a cystotome or capsule forceps, and a tortoise-shell scoop, generally fixed at the other end of the cystotome, as in Fig. 189). Further, in case they should be required, there should be at hand a caoutchouc or tortoise-shell spud for replacing the iris, and a wire vectis to remove the lens, should it become dislocated, or should there be any early escape of vitreous. It is useful to have also a wire elevator (Fig. 190), which can be used when the speculum has been removed, if it should be necessary, or appear advisable to remove that instrument before the completion of the operation.

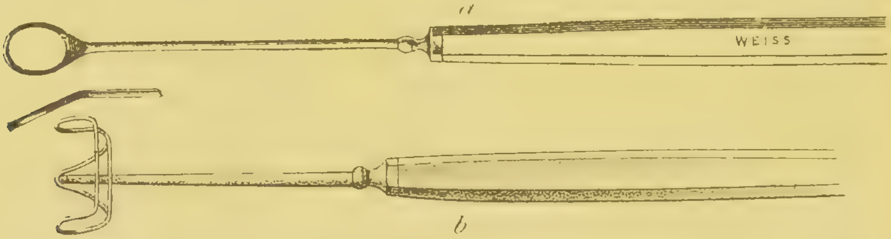

Fig. 190.—*a.* Wire vectis ; *b.* Wire elevator.

After the eye has been cocainised, and the conjunctival sac well washed out with corrosive sublimate solution, 1 to 5000, the speculum is introduced. The form of speculum shown in Fig. 188 is a useful one, as it can be used for either eye without getting in the way, and is sufficiently strong, owing to the rectangular shape of the arms, to resist any attempt on the part of the patient to close the eye. If the operator uses his right hand for making the section, he will stand behind the patient when operating on the right eye, and in front of him when operating on the left. This is necessary, as, owing to the nose getting in the way, the section has to be made from the temporal side.

If the section be made upwards, which position, though not always the easiest, on account of the tendency that there is to roll the eyes upwards, is for other reasons the best, the surgeon will cut towards him in operating on the right eye, and away from him in operating on the left. Either way is equally easy, but many operators always cut towards them, and make the section in the left eye with the left hand. It is rare indeed that this can be done with the same degree of precision, as a constant rule, as may be acquired with the right hand, unless the operator be really left-handed. In many cases a disproportionate amount of practice is required even to make a tolerably good left-handed operator, practice which is hardly justifiable for what is after all an unnecessary accomplishment. Of numerous operators whom I have seen, I have never yet seen one who could honestly be said to use either hand with equal facility, and on this account it is perhaps not too much to say that the patient should always be allowed the benefit of the best hand.

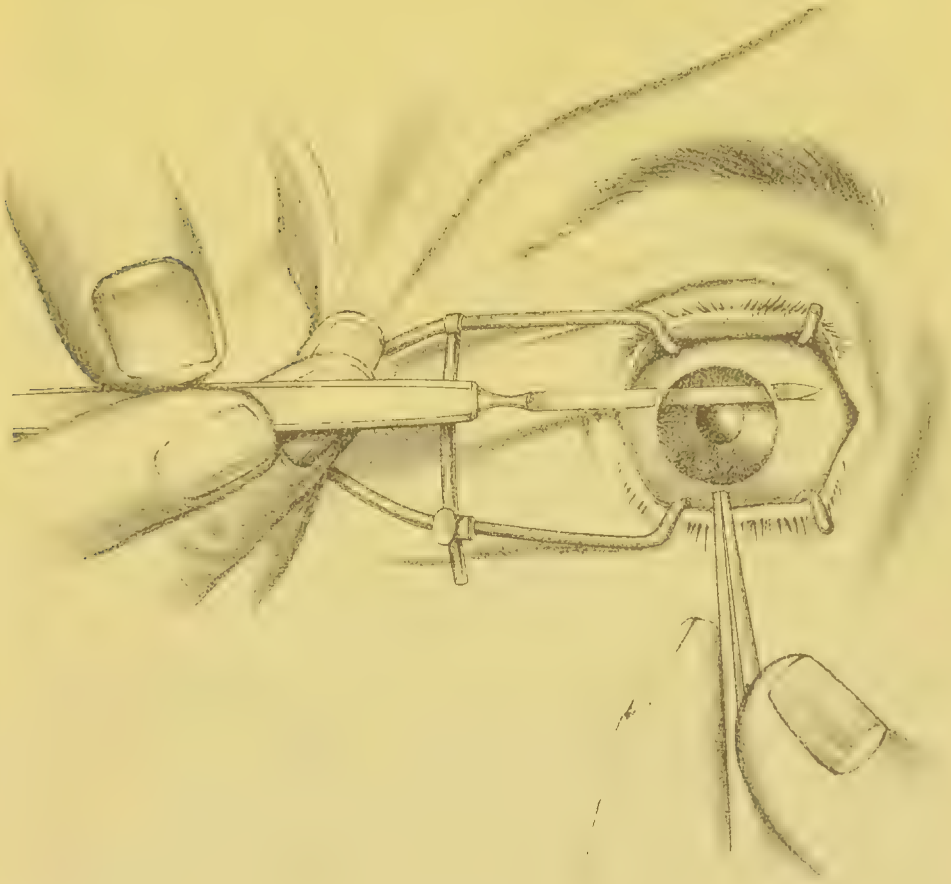

Fig. 191.

A firm hold is taken of the conjunctiva and subconjunctival tissue with the forceps held in the left hand. This fixation should be made in a line with the vertical diameter through the cornea, and therefore exactly opposite the mid-point of the section about to be made. If fixation be made at any other place, it may interfere with the proper performance of the section. The knife is then entered by making a *puncture* at the corneo-scleral margin, and at a point on a level with a semi-dilated pupil. In making the puncture, the point of the knife is directed towards the centre of the pupil, or rather lower. As soon as it has properly entered the chamber, it is steadily pushed forwards, while being gradually given a direction parallel, or nearly so, with the horizontal diameter through the cornea, until its point catches in the angle of the anterior chamber exactly on a level with the first puncture. At this point the *counter puncture* is made, and then the knife is quickly, and with as little sawing as possible, cut out, so as to make a section which lies throughout just about in the apparent corneo-scleral margin (see Fig. 191).

If the knife should catch too soon, that is, before its point has reached fairly to the angle of the chamber, it may be slightly withdrawn and directed towards a better point. This must be done carefully, and without increasing the size of the opening at the puncture, so as to retain the aqueous humour as far as possible. Sometimes, owing to the aqueous escaping, the iris falls in front of the knife immediately after the counter puncture has been made. When this happens two courses are open to the operator,—either to withdraw the knife slowly, and postpone the operation for some days, or proceed with the section, cutting through the iris at the same time. The latter does not interfere altogether with the successful termination of the operation, although it may complicate it by causing bleeding into the anterior chamber.

After the section has been made, if there be any little flap of conjunctiva, it should be turned over on to the cornea, so as to free the lips of the wound. A piece of iris is then removed with the iris forceps and scissors. A small iridectomy, 4 to 5 millimetres in width, is all that is required; it is therefore not necessary to exert any traction with the forceps, care being taken only that the pupillary margin is seized and drawn out. The cutting should therefore also be made with one snip, and not as was at one time very much practised, by means of several snips, freeing first the one side, and dragging out as much iris as could be got. By cutting the iris in this manner, and by making the incision in the way described, and not more peripherally, as was formerly done, we may to a great extent avoid any encleisis of the iris. After the iridectomy has been completed, it is well to see that the iris is free. If not, a little friction made with the lid over the eye will generally be sufficient to free it; but should this not be the case, recourse may be had to the caoutchouc spud, which can be used to much greater effect at this stage of the operation than after the lens has been removed. If there is now any manifest pressure of the speculum on the eye, or if the patient be unruly, that instrument should be removed, or an assistant may be allowed to hold it in such a way as to avoid any pressure. The cystotome is then introduced into the anterior chamber, the cutting edge or pricking point being held parallel with the surface of the lens, until it has been pushed as far down as the lower margin of the pupil, or, if it can be done without any difficulty, even behind the iris in this situation. The point is then directed to the lens, the capsule of which it readily pierces, and in which it is made to tear an opening by being steadily withdrawn in a vertical direction towards the external incision. A similar rent is then made in the capsule at right angles to this one, and as nearly as may be along its upper circumference. In this way an irregular ⊤-shaped opening in the capsule is obtained.

The object of opening the capsule is to permit of the easy escape of the lens. It is a point which has always been much discussed, which is the best way of doing this? I am not prepared to say that the way just described is better than any other that may be employed, but what is pretty certain is, that the more free the opening can be made, the more easily and completely can the opaque lens be removed. Care should therefore be taken that the cystotome really cuts the capsule, and is not allowed to pass in between it and the lens after having made a rent in it. It is to avoid the possibility of this that it is well to pass it far down first, and then to cut towards the periphery. A good opening can be got by using a pair of capsule forceps instead of a cystotome. The forceps are passed in closed, until the points are slightly beyond the centre of the lens, when they are opened, pressed gently against the surface of the capsule, and closed. They are then withdrawn slowly and by a slight side-to-side movement. In order to see whether they have removed a satisfactory portion of the capsule, they may be transferred at once to the dish containing the antiseptic lotion, and the portion of capsule allowed to float off. The capsule forceps are somewhat more difficult to manipulate than the cystotome. Care must be taken, of course, not to press too firmly on the lens, as it may readily in this way be dislocated. They must also be held in such a way as to avoid their catching further back from the points in any of the tissues in the external incision which would much interfere with its tearing away a portion of the capsule. Where a cataract is complicated with an opacity in the capsule, the forceps are far more satisfactory than the cystotome, as by using them the opacity is removed at the same time that the capsule is properly opened. This proceeding is safer than removing the capsular cataract after the lens has been extracted.

The next step in the operation is to effect the removal of the lens. For this purpose external pressure has to be made. The back of the tortoise-shell scoop is applied to the lower part of the cornea, and pretty firm pressure exerted. This causes the wound to gape, and the upper circumference of the lens to become engaged in it (see Fig. 192). When the lens has properly presented in this way the pressure is increased, and at the same time the scoop gradually caused to follow it upwards as it becomes more and more disengaged, until it is finally completely expelled. After this has been accomplished, it is well to wash out the sac again with the antiseptic lotion, and then, by pressing and rubbing up the lower lid against the cornea with the thumb, any cortical matter which has been rubbed off and remains in the eye is to be, as far as possible, got rid of. During this coaxing out of the cortical matter, care should be taken to keep the eye well washed with the corrosive sublimate

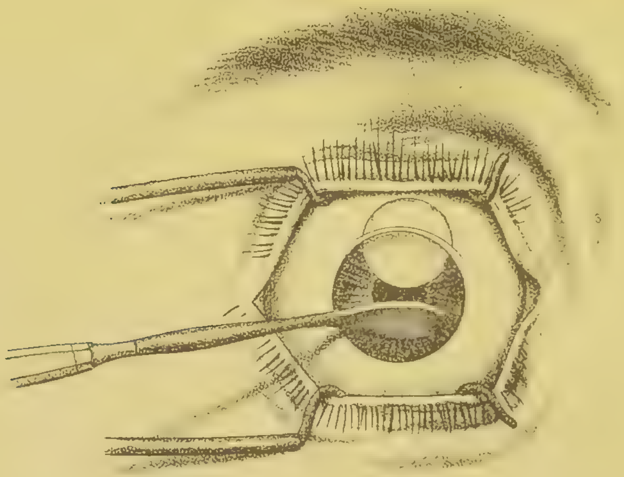

Fig. 192.

lotion, as it is otherwise not free from danger, owing to the possibility of micro-organisms being carried into the wound from the lid. It is better, however, except where there is dacryocystitis or chronic conjunctivitis, to make the necessary pressure on the cornea through the lid, and not directly, as it can be done with less irritation or injury to the cornea.

An idea of the completeness of the removal of the cortex may be got by seeing whether the patient can count fingers or not before applying the dressing. If this can readily be done, it shows that the pupil is clear. If the operation has been performed for an immature cataract, there may be some clear cortex remaining, but all opaque matter at all events has been removed from the line of vision. When this clearing has been made to a sufficient extent, or as far as may be advisable in any particular case, the caoutchouc spud should be run along the wound, in case any piece of capsule or cortex should be caught in it. If there should be any clotted blood, too, it must be removed with the iris forceps, and attention must be paid to the pupil, and any catching at the angles of the wound rectified in the manner already described. A dressing must be then applied.

Different dressings are used by different surgeons. The following may be recommended :—Next the eye, which is gently closed, is placed a piece of lint soaked in the corrosive sublimate solution, and on the top of this a piece of gutta-percha tissue of or about the same size, but not much bigger than the lint. A pad of absorbent cotton wool is then put on the top of this deep dressing, and kept in position with several turns of a flannel or domet bandage, or a couple of strips of adhesive plaster. This dressing is not changed for twenty-four hours, and after the eye has been examined, and a little corrosive sublimate lotion squeezed into the conjunctival sac, is reapplied as before, with the exception of the gutta-percha tissue. The same dressing is continued, and changed every twenty-four hours, for from five to eight days, after which all that is required is a light handkerchief and shade. If, as is generally the case, the chamber has re-formed within the first twenty-four hours, a dry dressing may be used on the third day, a little soft boracic or iodoform ointment being first applied to the margin of the lids to prevent the retention of the secretions. The advantage of the gutta-percha tissue at the first dressing is to keep the lint wet, and thus prevent any interference with the healing process from the accumulation of tears in the eye. It is not necessary to tie up the other eye, as the patient himself does not move either eye much, owing to the pain which this causes, until the anterior chamber has re-formed. The wound has generally so far healed by the time the first dressing is changed, so as to have permitted the chamber to become re-established. If there should be no chamber after two days—an unusual though occasional occurrence—it is better to remove the bandage, and replace it by a light one which exerts no pressure at all upon the eye. The patient should be kept in bed for a couple of days at any rate, unless he should be very corpulent, or should suffer from diabetes, when it is generally inadvisable to keep him in bed after the first day. For the first few days it is as well to keep the patient in semi-darkness, where this is possible; he should at any rate be protected from any strong light falling directly on the eyes.

If during the attempt to force the lens through the external wound any of the vitreous should escape, it becomes necessary to abandon the pressure on the cornea, as this would only cause further loss of vitreous without resulting in the escape of the lens. The lens has then to be extracted with the vectis, see Fig. 190 *a*. This instrument is pushed well behind it, by first being passed backwards, taking care not to dislocate it any further. When it has been got well in position behind the lens, it is withdrawn slowly, a slight pressure forwards being exerted all the time, so as to prevent the lens from slipping off by supporting it against the cornea in front. It is generally necessary to leave most of the cortical matter which may not have been extracted with the vectis. Some of it may be removed by carefully introducing the curette, but this proceeding should not be repeated too often, and if there has been a good deal of vitreous expelled at the same time as the lens, should not be tried at all. When there has been an escape of vitreous, extra care must be taken in the after treatment. It is better to tie up both eyes, and not to open the one on which the operation has been performed until after forty-eight hours.

Syringing out of the anterior chamber is recommended by M'Keown in cases where it is difficult to remove cortical matter, as where the cataract has been immature at the time of operation. This is used by him instead of the external pressure exerted either directly on the cornea or through the lid, as has just been described. This method does not seem to have had very many followers, and in point of fact it is seldom that, when the capsule has been properly opened, and some time is given for a little aqueous humour to accumulate, any such proceeding is called for.

When an extraction has been performed in an eye where there has been any dacryocystitis or conjunctivitis, it is a good plan to cover up the wound with a thick layer of finely powdered iodoform before tying up the eye. Such eyes are better left without a bandage as soon as the anterior chamber has re-formed.

In a very considerable proportion of cases of extraction some degree of iritis takes place during the healing process. As a rule this is very slight, but wherever there is any indication of it, atropine should be used to prevent any adhesion to the lens capsule. The most unfortunate accident which may happen after an extraction is suppuration of the corneal wound. If this goes on, it leads to more or less complete destruction of the cornea by extension of the inflammatory changes, and this, as well as a complication with purulent iritis, to which there is a tendency, results in the loss of all useful vision, and not infrequently in the complete disorganisation of the eye. This accident is probably always due to septic inoculation of the wound. It generally begins, so far as it can be observed, after the first twenty-four hours. The chamber is then found to be empty, the conjunctiva reddened and chemotic; the edges of the wound are slightly infiltrated, and a greyish haze extends more or less distinctly down into the cornea. The patient complains of pain and has often suffered during the night from sickness. When this state of matters is observed, no time should be lost in attempting to check the progress of the infiltration. This may be done either by using the thermocautery, or by applying a solution of nitrate of silver, 10 grains to the ounce, directly to the wound. The eye should afterwards be frequently bathed with the corrosive sublimate solution, or, better still, with freshly prepared chlorine water. Since the introduction of corrosive sublimate this accident is fortunately very much rarer than it formerly was. Occasionally it may be checked in the way described, but more often all attempts to do so fail.

In some cases of cataract it is advisable, instead of rupturing the capsule of the lens, to extract it as well along with the lens. Some operators even make this a rule. The result, as far as vision goes, is more immediately brilliant than when the capsule is left in the eye, but the operation is certainly much more risky, as it entails the loss of more or less vitreous, besides the irritation which may result from the tearing away of the suspensory ligament from its attachments. In all cases, however, where the lens is more or less dislocated, or where it is over ripe and shrunken, and also where it has undergone calcareous degeneration, it should be extracted in its capsule. Pagenstecher's curette or spoon (Fig. 193) should be used for this purpose. It is introduced well behind the lens, which by it is pushed up against the back of the cornea; a tortoise-shell scoop is then used, with which pressure is exerted on the cornea, so as to cause the lens to glide slightly upwards on the large spoon. When it is found to move, the spoon is slowly withdrawn, whilst at the same time external pressure is kept up with the scoop, which is made to follow the other instrument as it is removed. This operation requires the aid of a skilled assistant to manipulate the external scoop, while the operator holds the fixation forceps in one hand and Pagenstecher's spoon in the other. When carefully performed there need not be any great loss of vitreous; but that will depend to some extent on the consistency of the vitreous.

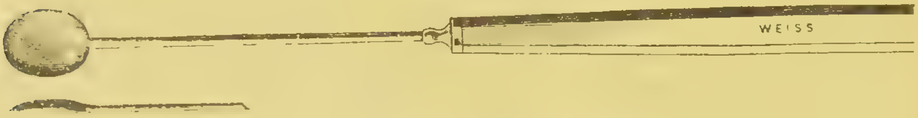

FIG. 193.—Pagenstecher's spoon.

In cases of capsular cataract, and of shrivelled-up congenital cataract in children, the whole thickened opaque membrane may be removed with the capsule forceps through a fair-sized linear incision. This operation does not entail any particular risks if it be done carefully. The capsule forceps having been introduced, and a good hold of the membrane having been got with them, they must be very slowly removed, and with a slight side-to-side movement. If it is found that great resistance is offered to the complete removal, as much as readily comes away may be snipped off with the iris scissors, and the rest allowed to be dragged back again into the eye.

If, owing to having made too small an incision, or to any other cause, considerable difficulty has been experienced in extracting a cataract, so that the scoop has had to be used more freely on the cornea than usual, there will be found on examining the eye a milky opacity of the cornea, which, on close inspection, will be seen to be due to a finely striated interstitial opacity, the result no doubt of some stagnation in the lymph channels. This appearance may remain for a number of days, but eventually clears off. It is due to injury to the cells lining the back of the cornea, and is favoured by the action of corrosive sublimate when some of the lotion used during the operation passes into the chamber.

An opacity due to cocaine has been described, but this I have never seen, though I have all along used it freely, and in combination with corrosive sublimate and different other antiseptics, as well as with mydriatics and myotics. There can be little doubt that this is due to the presence in some specimens of cocaine of an irritant alkaloid which cannot readily be separated from the cocaine. The opacity produced appears to be confined to the epithelial layer of the cornea, though it may nevertheless remain permanent.

A number of operators, and amongst them some of the most experienced, have recently reverted to the old plan of extracting without iridectomy. It is not difficult to understand that those who were in the habit of making very large iridectomies, such as were the rule for some time after the introduction of the modified linear extraction, should have resorted to the simple operation and discarded iridectomy altogether. These large iridectomies are certainly unsightly, and the proportion of cases in which encleisis takes place, when the section is made as peripheral as von Graefe recommended, is by no means trifling. Besides, the large peripheral portion of the cornea, through which rays are thus permitted to reach the retina, is apt to render the image less sharp. On the other hand, the same disadvantages do not attend the performance of a small iridectomy and a less peripheral incision. There is little tendency to bad encleisis and little interference with vision, especially when the iridectomy is done upwards. The removal of the cataract is also easier if unripe, and the risks on subsequent bursting of the wound are less. The only disadvantages are, therefore, a wound in the iris and a less beautiful pupil. Those who have been in the habit of operating in this manner may fairly claim that the former disadvantage, which may even be got rid of altogether by the performance of a preliminary iridectomy, is at least outweighed by the diminished risk of encleisis or bad prolapse of iris in case of the wound bursting where no iridectomy has been done. The cosmetic advantages of a round active pupil, when it can be obtained, are altogether trifling in those elderly individuals who are the usual subjects of cataract extraction. The game is, in fact, not worth the candle.

OPERATIONS FOR AFTER CATARACT.

In a certain proportion of cases in which a cataractous lens has been removed, it becomes necessary, sooner or later, to perform some operation in order to further increase the transparency of the pupil. The proportion of cases where this is necessary will be less, according as one has at the time of operation been successful in removing the cortical lens matter from the eye, or according as the subsequent absorption of any remaining cortical matter has been more and more complete. The degree of iritis, too, following the extraction is of influence in this respect. When there has been any considerable degree, a subsequent operation will always be necessary before good vision can be obtained. The operation to be selected in any case depends upon the degree of opacity as well as upon the nature of the membrane.

The Double Needle or Tearing Operation.—Some time previous to performing the operation, atropine is dropped into the conjunctival sac, in order to get as great a retraction of the iris as possible. When the speculum has been introduced, the operator, standing behind the patient's head, enters a stop needle with his right hand through the cornea at a short distance from its apparent margin. If the right eye be the one operated on, the needle is entered at the outer side, if the left at the inner side. It is pushed obliquely inwards so as to pierce the membrane occupying the pupil at a point opposite the centre of the cornea. Holding the needle steadily in position, a similar one is next introduced with the left hand through a corresponding and opposite point of the cornea, and pushed through the membrane alongside of the first; then, by simultaneously raising the handles of both needles, their points are made to describe circles in opposite directions round the portions where the cornea is penetrated, and a hole is thus lacerated in the membrane. The needles should not be pushed further through the membrane than just sufficient to pierce it. After a good hole has been got in this way they are quickly withdrawn and a dressing applied for twenty-four hours, in the same manner as has been described as suitable after extraction.

This operation is best suited for obtaining an opening in very fine membranes, such as are composed almost entirely of the opaque capsule, and which have gradually developed after a longer or shorter period of greater transparency.

The Narrow Knife or Cutting Operation.—In this operation a knife about two-thirds as long and one-half as broad as an ordinary Graefe's knife is used. The operator steadies the eye with the fixation forceps, and introduces the knife through the outer or inner side of the cornea, according as the right or left eye is the one operated on. The cutting edge is directed towards the eye, and the knife pushed obliquely inwards until its point penetrates the membrane as far over to the opposite side as is possible without wounding the iris. It is allowed to remain in this position for a second, and then in raising the handle the blade is made to cut its way through the membrane.

In performing this operation, it is important not to make an attempt to sweep round the blade immediately after it has entered far enough into the chamber, as by so doing one frequently fails, even with a very sharp knife, to obtain a good opening, owing to the yielding nature of the membrane and its attachments. By first piercing the membrane, however, as described, and then waiting for a second or two, it comes to lie with its cut edge up against the edge of the knife, and is easily divided without any traction. This is the best operation in all cases where the membrane is at all dense. Not only is a better opening obtained in this way than with needles, but it gives rise to no irritation. It is suitable in all cases where an operation is required shortly after extraction to complete the transparency of the pupil, and where there has not been so much iritis as to lead to any drawing up of the pupil. The double needle operation is often used for such cases as well; but when the membrane is tough it is more difficult to procure in this way a satisfactory opening, a longer manipulation is necessary, and, besides this, a degree of dragging on the attachments of the membrane is caused, which may lead to very considerable irritation, and the eventual closing up of the aperture. When the operator has assistance the cutting operation may be performed by oblique illumination with the patient seated on a chair in the dark room.

The Scissors Operation, or Iridotomy.—A good hold is taken of the conjunctiva with the fixation forceps, and a narrow keratome introduced into the anterior chamber as near as possible to the corneo-scleral margin. The keratome may be made to pierce the iris at the same time. The incision should not be too small, as it should be large enough to enable one to use the iridotomy scissors freely; it should therefore be at least

3 millimetres at its inner opening. After the keratome has been slowly withdrawn, a pair of Wecker's iridotomy scissors are introduced. The one blade, which should preferably be sharp, is passed well underneath the iris and membrane, and the other above it until the opposite angle of the chamber is reached, when, by a firm snip, the intervening tissues are divided, and the scissors quickly withdrawn closed. The direction in which the blades of the scissors are passed should be at right angles to the stretched fibres of the iris.

This operation is suitable for most cases where the pupil has become closed and drawn up owing to iritis after extraction. The iridotomy scissors are often made too long in the blades. They should not be much larger than two-thirds the diameter of the anterior chamber or three-eighths of an inch. The difficulty in performing the operation properly is to get the blades sufficiently separated so as to pierce through the whole thickness of the obstructing screen near enough to the side at which the section is made. It is on this account that it is advisable not to make the section too small, and at the same time to pierce the iris and subjacent membrane with the keratome.

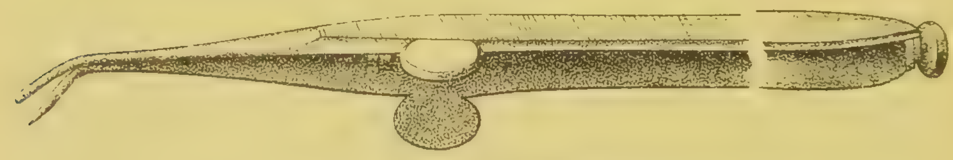

FIG. 194.—Iridotomy scissors.

Excision of a triangular piece of Iris.—In cases where the iritis following extraction has been very severe, and where there is consequently a want of elasticity in the opaque screen, owing partly to wasting of the muscular tissue of the iris, and partly to the tough nature of the plastic tissue to which it is firmly glued, the following proceeding may sometimes give a fair result:—A small flap consisting of both cornea and iris is made about midway between the centre and lower circumference of the cornea, but rather nearer the latter. Two converging snips upwards are then made with the scissors from either end of the iris flap, and thus a triangular piece of iris and subjacent membrane isolated, which is then seized and removed with the iris or capsule forceps. In these cases the vitreous is often very fluid, and a considerable amount necessarily escapes during the operation, the permanent effect of which depends upon the degree of reaction following, as well as upon the clearness of the vitreous.

STRABISMUS OPERATIONS.

Two operations are practised for the cure of strabismus. If, for instance, the degree of convergence be in excess of that required for binocular fixation, we may have to divide one or both internal recti, *i.e.*, perform a *tenotomy*, or we may have to bring forward the tendon of one or both external recti, *i.e.*, perform an *advancement* of the muscle.

There are various methods in use of performing both tenotomy and advancement. The particular cases for which these operations are suitable, and the precautions to be taken in connection with them, have already been referred to in Chapter XVII.

Tenotomy is now always performed by cutting across the tendon of the muscle close to its insertion to the sclera, according to the method introduced by von Graefe. This permits of a certain degree of retraction, but the presence of other indirect attachments prevents the retraction being too great.

The instruments required for the operation (see Fig. 195) are a speculum, or a couple of Desmarres' elevators, fixation forceps, a pair of blunt-pointed scissors (curved on the flat), and a strabismus hook. A tenotomy can generally be performed without general anæsthesia. A drop or two of a 5 per cent. solution of cocaine produces sufficient local anæsthesia, as a rule, and it is better, in order to be able to judge of the effect of the operation, that the patient should be fully awake. In performing tenotomy of the internal rectus of the right eye, or the external rectus of the left eye, the surgeon stands behind the patient's head. For the other lateral muscles he may stand in front at either side—preferably the left. After having inserted the speculum, a hold is taken with the fixation forceps of a piece of conjunctiva lying over the insertion of the muscle, a good quarter of an inch therefore from the border of the cornea. A vertical snip is next made with the scissors large enough to allow the blades to be opened pretty freely underneath the conjunctiva. The scissors are then made to cut their way backwards immediately underneath the conjunctiva until they cease to encounter any resistance, and one is able to feel that their points can be freely moved about. The scissors are then withdrawn, and the hook inserted underneath the muscle. This is done by first passing it backwards along the upper or lower edge of the muscle, according to the one operated on, in such a way that its horizontal and convex portion is parallel with that edge. On then turning it quickly round it slips below the muscle and is drawn forwards until it is arrested by the attachment of the tendon.

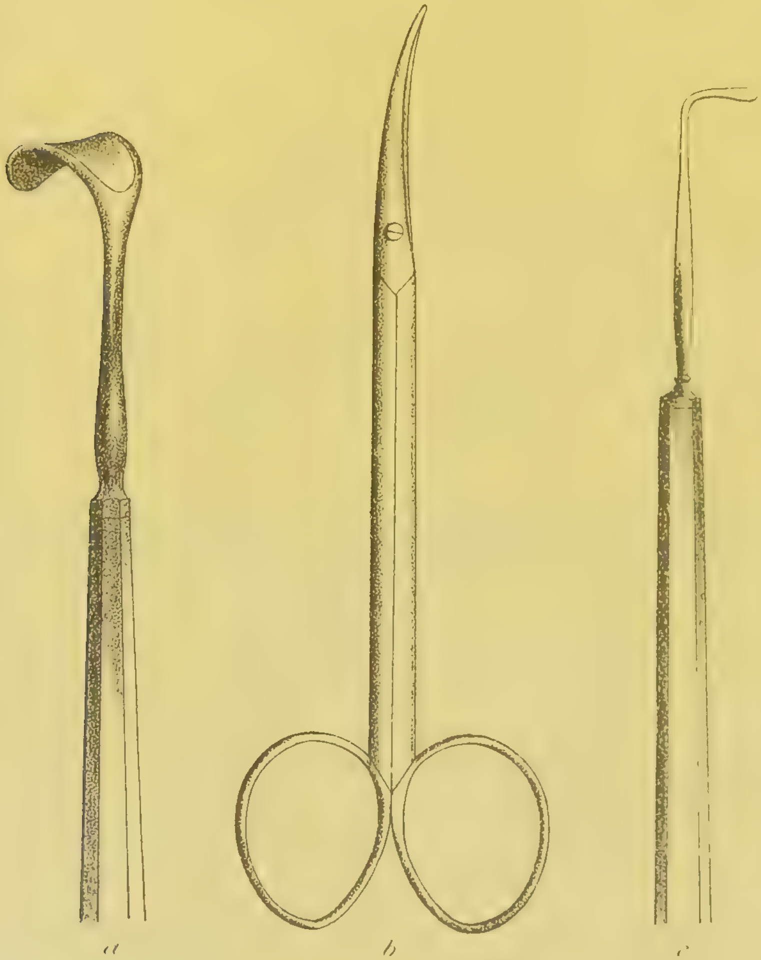

FIG. 195.—*a.* Desmarres' elevator ; *b.* Strabismus scissors ; *c.* Strabismus hook.

It is then transferred to the left hand, and the tendon cut between the hook and the eye. After this has been done the hook is again inserted and swept upwards and downwards with the object of testing whether the division has been complete. If it should catch on any portion of the insertion which has escaped division, this portion must be divided with the scissors. A suture is then used to bring the edges of the wound in the conjunctiva together. The suture should be passed *diagonally* *downwards and inwards*, and only take in the conjunctiva. A deeper suture placed horizontally is sometimes required if the effect of a tenotomy has been too great. The eye should afterwards be bathed occasionally with corrosive sublimate lotion, but no bandage is required.

This method of performing tenotomy, which is that formerly practised by von Graefe, only very slightly modified, is the best, as the effect produced is not only greatest, but admits to some extent of being regulated by sutures. Though not perhaps quite so easy as the subconjunctival method, the advantage over that operation is that the operator is able to see exactly what he does. The introduction of a suture also renders sinking of the caruncle less likely to occur.

Subconjunctival Tenotomy.—The operator stands in front of and to the right side of the patient. The same instruments are used, with the exception of the scissors, which in this operation are straight. A snip is made with them in the conjunctiva at a point slightly below the level of the lower border of the insertion of the muscle. The deeper subconjunctival tissues are then seized with the forceps and the opening extended into them. When this has been done the strabismus hook is slipped in and slightly swept round so as to catch under the muscle. It is then transferred to the other hand and held up while the scissors held in the right hand are passed in through the external opening, with one blade in front of and the other behind the insertion of the muscle which the hook thus puts on the stretch. The insertion is then cut across with one or two snips of the scissors. If this has been completely done the hook can then be passed forwards until it catches on the tissues immediately surrounding the cornea. No stitch is required.

Snellen's method of performing tenotomy is as follows:—A horizontal incision is made in the conjunctiva over the middle of the tendon of insertion of the muscle. A portion of the tendon is then seized with the forceps and a snip made into it with a pair of scissors, which for this purpose may be either straight or curved. A small strabismus hook is then passed in under the upper portion of the tendon through this hole, and this portion divided with the scissors, one blade of which is passed behind and the other in front of the hook. The lower half of the tendon is then divided in the same way close to its insertion.

Snellen claims for this method of operating that the direct insertion is separated without any interference with the indirect insertions, or with the capsule of Tenon, so that the retraction takes place equally.

OPERATIONS FOR ADVANCEMENT OF A RECTUS MUSCLE.

A number of methods are also in use for the advancement of the internal or external rectus. Of these three may be referred to.

1. A vertical incision is made in the conjunctiva over the muscle to be advanced, and of a length equal to the full breadth of the muscle. A thread is then passed round the muscle close to its insertion by catching it up with a strabismus hook having an eye at the end through which the thread is threaded. After this thread has been firmly knotted round the tendon the ends are left long and the insertion of the muscle divided close to the sclera. Keeping hold of the thread in one hand, the operator proceeds to free the muscle from its attachments all round, above, below, and to either side for some distance back. He then introduces a double thread through the muscle from its under surface. The thread used for this purpose should be of pretty stout waxed silk, with a needle at the middle and one at either end. The middle needle is passed through the muscle. Having seen that this has been properly done, and that the freed and retracted muscle can be readily drawn forwards by the double thread, the first thread round the tendon, by which it was held, is cut away, together with a portion of the end of the muscle. One of the needles at the end of the double thread is then passed underneath the conjunctiva, and brought out at a point close to the vertical meridian through the cornea, while the other is passed in the same way beneath the conjunctiva below, and brought out at a point opposite the first. The middle needle may then be cut off, leaving two threads instead of one. The ends of the two threads are then tightly knotted, and thus a traction on the muscle exerted which draws it forwards, the one thread drawing it upwards and forwards, and the other downwards and forwards, so that the resultant, if they are properly applied, is to advance the muscle in the meridian of its action. Finally, a stitch is put in to keep the edges of the conjunctival wound in contact.

This operation is essentially von Graefe's modification of Critchett's method of performing advancement. It is well to remove a portion of conjunctiva before knotting the threads. Instead, too, of the single thread, two may be used, each one penetrating a different portion of the muscle. The amount of effect which can be got by it depends on the position at which the muscle is perforated by the thread, and partly as well on the tightness with which the threads are drawn and on the size of the piece of tendon removed.

2. *Schweigger's method.*—In this operation catgut stitches are used instead of silk. After a vertical incision has been made in the conjunctiva over the muscle, the muscle is freed above and to either side, and a catgut suture passed round it at some distance from its insertion and firmly knotted on it, the ends of the suture being left long. Another catgut suture is passed through the muscle behind the first. The insertion is next divided and a portion of the end of the tendon cut off. The free ends of the catgut sutures are then introduced under the conjunctiva, the ends of the first being entered close together and in a line with the muscle, and the other two ends above and below respectively. The two uppermost are then knotted together, and also the two lowermost. The first suture is placed round the muscle to prevent the second cutting its way through it. To obtain the greatest effect, the sutures must be placed as far back as possible.

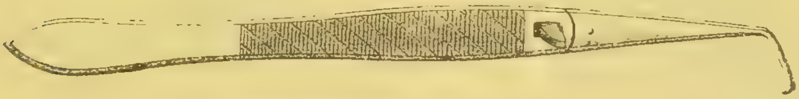

Fig. 196.—Prince's advancement forceps.

3. An operation differing considerably from these just described was introduced a few years ago by Prince. To this he gave the name of *pulley operation,* owing to the manner to which the advancement is effected. The method in which he performs the operation is thus described by him:—"The eye being fixed, the anchor or pulley suture *a* is introduced slightly into the dense tissue, one millimetre from the corneal margin. The conjunctiva and capsule of Tenon having been divided, one branch of the advancement forceps (Fig. 196) is introduced underneath the tendon of the rectus, and the other closed upon it,

securing the edge of the retracted conjunctiva, after which the tendon is separated from the sclera. Each end of a thread being armed with a needle, both are passed from underneath the elevated rectus through the capsule, muscle, and conjunctiva, enclosing the middle portion of the rectus in a loop, from which it cannot escape. The tissues in the grasp of the forceps are now divided two millimetres anterior to the loop suture, the location of which will depend on the amount of advancement required in each individual case. One end of suture *b* is curved over suture *a*, both ends of which are now brought together and securely tied, enclosing the former in a loop or pulley. Both ends of *b* are now brought together in the form of a surgical knot, and it becomes apparent that in proportion as they are tightened over the pulley formed by *a*, will the cut end of the rectus be advanced, simultaneously closing the conjunctival gap. To obtain the most perfect correction, a bow-knot is applied and time allowed for recovering from the effect of traction, which should as much as possible be avoided during the operation. Afterwards this knot may be secured, or the effect increased or diminished as conditions may indicate."

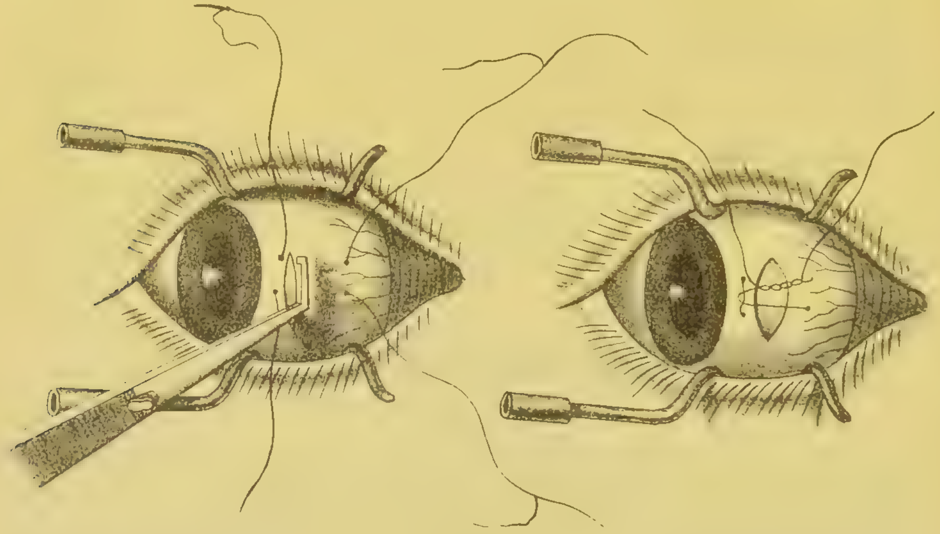

FIG. 197.—(After Prince.)

I have found this a very simple and efficient operation. The manner in which I have performed it, though the same in principle, differs slightly in details. The following are the points of difference. The pulley suture is introduced by running it out and in two or three times, over a larger extent of the circumcorneal connective tissue, which gives it a better hold, as it has to bear a considerable strain and should not readily cut its way through the tissues. The other suture is passed single instead of double, and through the muscle rather further back than through the conjunctiva. The muscle has therefore to be freed first to some extent. The portion of tendon grasped by the advancement hook is cut off. The bow-knot is discarded altogether, as not having any particular value, but the operation is done without an anæsthetic, and the thread pulled sufficiently tight to produce at the time a slight exaggeration of the effect required.

EVISCERATION OF THE GLOBE.

The instruments required for this operation are a speculum and fixation forceps, a Graefe's knife, a pair of scissors, and a blunt Volkmann's spoon. The conjunctiva is first undermined for a short distance all round the cornea. The anterior chamber is then transfixed on a level with the horizontal meridian of the cornea, and a section made which completely separates the lower portion of the cornea from the sclera along the junction between them. The flap of cornea thus formed is then seized with the forceps, and the rest of the cornea separated with the scissors. With the Volkmann's spoon the whole contents of the globe can now be evacuated. This must be done thoroughly, so that nothing is left but the sclera. When the bleeding which this causes has been stopped by means of pledgets of cotton wool introduced into the cavity, the edges of the conjunctiva are brought together with sutures. The sclera should not be stitched.

This operation is always followed by considerable œdematous infiltration of the tissues, and pain, which lasts at least twenty-four, often forty-eight or more hours. I have not found that the introduction of horse-hair or catgut as a drain has modified the severity of the infiltration, although it has been recommended for this purpose. The stump left after this operation, though admitting of a better movement in an artificial eye, when it can be fitted in to the orbit, than results from enucleation of the eye altogether, is yet not so markedly better in this respect as might be supposed from the appearance shortly after operation. A very great degree of shrinking takes place in the course of time, so that the pad on which the muscles act is greatly reduced in size. To obviate this defect, a modification of the operation was recommended by Mules, which consists in introducing a glass ball into the scleral cavity and uniting the conjunctiva over this. I have no personal experience of Mules' operation, but understand that it has been fairly successful in his hands. In a great many cases the glass ball seems to be expelled sooner or later by ulceration of the over-lying tissues, but the eventual result in such cases does not appear to be worse than if the attempt had not been made, although the patient is necessarily subjected to a somewhat prolonged treatment. When the operation succeeds, the effect, so far as the movements of the artificial eye, which rests on this artificial stump, go, could hardly be more beautiful.

Enucleation of the Eyeball.

The instruments required for enucleation are a speculum, fixation forceps, strabismus hook, and a pair of strong scissors curved on the flat. Anæsthetics should be used except when there is good reason for avoiding them, when the pain may be to a great extent mitigated by the free use of cocaine during the operation. The conjunctiva immediately surrounding the cornea is first incised. This is best done by catching hold of a portion of the upper part with the forceps, snipping through it with the scissors, and then passing one blade behind, allowing it to glide beneath the conjunctiva to one side of the cornea, while the other blade remains external to the conjunctiva. In this way one or two cuts with the scissors will suffice to complete the division of one-half of the conjunctiva surrounding the cornea, while the other half may be divided by using the blades in the same way along the other side after having begun again at the point above, where the first perforation was made. This having been done, the capsule of Tenon is opened by a more free use of the scissors at the lower portion, just over the inferior rectus. The strabismus hook is then passed under the inferior rectus, which is divided close to its insertion. The hook may now be swept in succession round each of the recti muscles, and their insertion severed from the globe in the same way. After dividing the insertion of the superior rectus, the hook should be passed deeper so as to hook up the superior oblique, which must also be divided in the same way. The branches of the speculum are then separated as much as possible, and the eye deprived of its muscular attachments allowed to protrude. The scissors must next be passed in between the capsule and the globe to the back of the eye, the blades being kept closed until the points are felt to touch the optic nerve. They are then opened and pushed a little further, so as to cause the blades to pass one on either side of the nerve, which is cut across with a firm snip. The eye can now be drawn forwards, as it is only held by a few bands of connective tissue, which have to be snipped across to complete its removal. The bleeding is readily stopped with pledgets of cotton wool. The proper arresting of the bleeding, as well as careful antiseptic treatment throughout, should be attended to, as these points are of importance in ensuring the speedy healing of the wound left in the orbit. A tight bandage should be applied and kept on for four or five hours, so as to prevent any infiltration of the tissues with blood. Afterwards nothing is required but attention to the cleanliness of the wound by frequent syringing or bathing with corrosive sublimate solution. It is a good plan, too, to smear a little lard along the edges of the lids at night, so as to prevent any retention of the secretions.

An artificial eye should not be worn for at least two months. The eye when worn should be removed every night, and kept in water containing a little antiseptic. When it becomes corroded it should be repolished, and at any time any irritation which it may produce should be taken by the patient as a warning that its use should be left off for some time. There is a great tendency amongst the wearers of artificial eyes to have them too big. They do not in looking at themselves in the glass appreciate the staring appearance of such an eye. It is better to have one of such a size that the lid droops slightly over it as compared with the other eye. Such an eye is not only less likely to set up irritation, but is also much more deceptive.

Operations are sometimes required to render the socket better fitted to lodge an artificial eye. No definite rules can be laid down for such operations. The surgeon must be guided by the conditions presented by each individual case. Sometimes, owing to the tendency to drooping of the lower lid, it is necessary to raise it by uniting the upper and lower lids at the outer canthus.

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